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American Medical Association. Section on Nervous and Mental Diseases, American Medical Association

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TRANSACTIONS OF THE

SECTION ON

Nervous and Mental Diseases

of the
American Medical Association
at the Sixty-Sixth Annual
Session, held at San Francisco,
June 22 to 25, 1915

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This Section has been in existence under its present name since 1900. The branches of the practice of medicine which comprise its province were classified with some other subjects in the early days, as is shown by the following section names. The list of officers has been compiled from the published records, which are not complete—for example, the city is not given in some cases. It will be appreciated if any additional information is brought to the attention of the Secretary of the American Medical Association.

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PROCEEDINGS OF THE SECTION

Tuesday, June 22—Afternoon

The meeting was called to order at 2:30 p. m., the Chairman, Dr. Francis X. Dercum, Philadelphia, presiding.

Dr. Francis X. Dercum, Philadelphia, read the Chairman's Address: "Recent Aspects of Nervous and Mental Pathology." No discussion.

The Chairman appointed Drs. William A. Jones, Minneapolis, George H. Moody, San Antonio, and Ross Moore, Los Angeles, members of the Executive Committee, and Dr. James H. McBride, Pasadena, Calif., Delegate to the House of Delegates.

The Chairman appointed the following Nominating Committee: Drs. Charles R. Ball, St. Paul; H. C. McClenahan, San Francisco, and J. T. Fisher, Los Angeles.

Dr. Adolf Meyer, Baltimore, read a paper on "Objective Psychology and Psychopathology with Subordination of the Medically Useless Contrast of Mental and Physical." Discussed by Drs. Thomas J. Orbison, Los Angeles; Josephine A. Jackson, Pasadena, Calif.; Andrew L. Skoog, Kansas City, Mo.; Cecil E. Reynolds, Los Angeles, and Adolf Meyer, Baltimore.

Dr. Charles Lewis Allen, Los Angeles, read a paper on "Delusions of Persecution as a Biologic Defense Reaction." No discussion.

Dr. Mary Lawson Neff, Des Moines, Iowa, read a paper on "Serviceable Psychology." Discussed by Drs. Elizabeth Kearny, Los Angeles; Charles R. Ball, St. Paul; E. B. Hoag, Los Angeles; C. Eugene Riggs, St. Paul; W. J. G. Dawson, Eldridge, Calif.; H. L. Ewing, Lincoln, Neb.; C. L. Allen, Los Angeles, and Mary Lawson Neff, Des Moines, Iowa.

Dr. J. T. Fisher, Los Angeles, read a paper on "Traumatic Hysteria." Discussed by Drs. Thomas J. Orbison, Los Angeles; Charles L. Allen, Los Angeles; Walter F. Schaller, San Francisco; Andrew L. Skoog, Kansas City, Mo.; Charles R. Ball, St. Paul; Francis X. Dercum, Philadelphia, and J. T. Fisher, Los Angeles.

Thursday, June 24—Morning

The Nominating Committee offered the following report on nominations for the ensuing year: Chairman, Dr. George A. Moleen, Denver; Vice-Chairman, Dr. M. A. Bliss, St.

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Louis; Secretary, Dr. A. S. Hamilton, Minneapolis; Delegate, Dr. Milton B. Lennon, San Francisco; Alternate Delegate, Charles L. Allen, Los Angeles.

Approved on motion and carried.

Dr. W. A. Jones, Minneapolis, read a paper on "Myotonia Congenita." Discussed by Drs. Frank R. Starkey, Philadelphia; G. W. Robinson, Kansas City, Mo.; F. X. Dercum, Philadelphia, and W. A. Jones, Minneapolis.

Dr. J. P. Munroe, Charlotte, N. C., read a paper on "Chronic Tetany; Report of Case." Discussed by Drs. J. T. Fisher, Los Angeles; J. M. Wolfsohn, San Francisco; Cecil E. Reynolds, Los Angeles; Josephine A. Jackson, Pasadena, Calif.; Frank R. Starkey, Philadelphia; George A. Moleen, Denver, and J. P. Munroe, Charlotte, N. C.

The following papers were read as a symposium:

Dr. T. H. Weisenburg, Philadelphia, and Dr. Philip Work, Pueblo, Colo., (read by Dr. Work), "The Diagnosis of Tumors in the Posterior Cranial Fossa."

Dr. Ernest G. Grey, Boston: "Cerebellar Tumor Without Nystagmus."

Dr. I. Leon Meyers, Chicago: "Galvanometric Studies of the Cerebellar Function."

The above papers were discussed by Drs. A. L. Skoog, Kansas City, Mo.; G. W. Robinson, Kansas City, Mo.; John S. Turner, Dallas, Tex.; Cecil E. Reynolds, Los Angeles; George A. Moleen, Denver; Philip Work, Pueblo, Colo.; W. F. Schaller, San Francisco; Ernest Grey, Boston, and I. L. Meyers, Chicago.

Dr. Charles R. Ball, St. Paul, read a paper on "Focal Disease of the Brain: Report of Eight Cases." Discussed by Drs. C. Eugene Riggs, St. Paul; Cecil E. Reynolds, Los Angeles; D. M. Hansen, Lincoln, Neb.; George A. Moleen, Denver, and Charles R. Ball, St. Paul.

Thursday, June 24—Afternoon

Dr. Benjamin T. Burley, Worcester, Mass., read a paper on "Acute Ascending Hemorrhagic Myelitis." Discussed by Drs. Charles R. Ball, St. Paul, and B. T. Burley, Worcester, Mass.

Dr. A. L. Skoog, Kansas City, Mo., read a paper on "Spinal Cord Compression from Leptomeningeal Cysts: Report of Two Cases." No discussion.

Dr. C. Eugene Riggs, St. Paul, read a paper on "Salvarsanized Serum in Syphilitic Nervous Disease." Discussed

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by Drs. G. W. Robinson, Kansas City, Mo.; C. R. Ball, St. Paul; Frank R. Starkey, Philadelphia; George H. Moody, San Antonio, Tex.; W. J. G. Dawson, Eldridge, Calif.; A. W. Hoisholt, Napa, Calif.; G. P. Sanborn, Boston; H. G. Brainerd, Los Angeles; Cecil E. Reynolds, Los Angeles; F. X. Dercum, Philadelphia; Philip Hillkowitz, Denver; W. A. Jones, Minneapolis; W. V. Brem, Los Angeles; George A. Moleen, Denver, and C. Eugene Riggs, St. Paul.

Dr. Ross Moore, Los Angeles, read a paper on "Testing Muscular Tonus or Reflex Excitability in the Upper Extremity." No discussion.

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RECENT ASPECTS OF NERVOUS AND MENTAL PATHOLOGY *

FRANCIS X. DERCUM, M.D.
PHILADELPHIA

The pathology of the nervous system has heretofore and in a large measure occupied a place by itself, that is, a place apart from pathology in general. Recent developments, however, have shown that only in a limited sense is this justified. Many of the organic diseases of the nervous system—some of its neoplasms and system diseases—must of necessity occupy a separate and distinct position, but is this true of the functional diseases and especially of those which are classified as mental? Are not the phenomena of the latter to be included in the general pathologic processes going on in the body as a whole? Let us see what the facts indicate.

First, the various functions of the human organism, digestion, nutrition, the maintenance of the bodily temperature, the production of nervous and muscular energy are all the expression of chemical processes; secondly, these chemical processes, manifold and complicated, are intimately correlated. It is the derangement of these processes that constitutes disease. Such derangement may arise spontaneously through some fault in the makeup of the individual, or it may be secondary to some disturbing cause introduced from without, such as a poison or infection.

Disturbances of the metabolism of the body, no matter how arising, necessarily entail states of intoxication and in these states of intoxication must be

* Chairman's Address.

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sought the explanation of nervous and mental disorders. Even many of the affections which we classify as organic, for instance, the system diseases, owe their inception to the presence of toxic substances, and the subsequent death of systems and tracts is to be looked on merely as a terminal state. It is, however, especially in the so-called functional nervous affections (and among these the mental affections are to be included), that the recognition of the factor of intoxication is important.

Time will not permit us to recall the various theories of toxicity that have from time to time appeared in the history of medicine. Such theories appear in Hippocrates, were advocated by Pinel, Esquirol and Morel and in more recent times by Mendel and by Bouchard and his school. Although numerous and interesting facts were discovered, the existing knowledge of physiologic and chemical processes was not sufficiently advanced to enable medical men to interpret them correctly or to assign them a proper value, and because the studies proved disappointing the field was for a time neglected. That poisons do play a rôle, however, in the production of nervous and mental disease became evident from the subsequent and rapidly accumulated knowledge as to the rôle played by poisons introduced from without, notably alcohol and the infections. In regard to alcohol it became known not only that it may bring about diseased states in various of the viscera, but also that it may bring about inflammation of the nerves, and, in addition, disturbed mental states consisting of delirium, confusion and stupor. As regards the infections, Regis and later Kraepelin pointed out that they also were responsible for both nervous and mental symptoms. Kraepelin was one of the first to insist that in infections mental symptoms are the result of the action of bacterial toxins. These toxins, it would appear, may act directly on the brain, or the infection may bring

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about disturbances in the function of other organs, disturbances leading to changes in the general metabolism of the body, to the formation of endogenous toxins and a consequent general autointoxication. Infections variously involve the liver, the kidneys, the thyroid, the adrenals and other glands and tissues. The fact of such involvement is based on indisputable clinical and pathologic evidence. That under such circumstances an intoxication may be very complicated and very persistent can be readily understood. The prolonged confusions that now and then occur during the convalescent periods of typhoid fever and other acute infections, the prolonged confusional insanity every now and then encountered in alcoholism, are clearly due not to the original infection, not to the poison originally ingested, but to the secondary toxemia of a deranged metabolism. The confusional insanity of alcoholism, for instance, lasts, as we all know, for many months after alcohol has ceased to be ingested, and there is no escape from the conclusion that in such cases the symptoms are due to secondary intoxications.

These ideas must necessarily influence our conceptions as to the nature and origin of primary autointoxication, that is, of endogenous poisoning. A moment's reflection reveals that primary autointoxication is of two kinds; first, one that is gastrointestinal in origin, and, second, one that arises in the tissues of the body itself. In the normal or healthy individual, intestinal poisons are destroyed by the liver, the thyroid and by other glands and by the tissues generally, or they are eliminated by the various excretory organs. Poisoning of the nervous system is thus effectively guarded against. In diseased and defective organisms, however, invasion may occur, and the nervous system may suffer both from this primary intoxication and from secondary disturbances of metabolism due to derangements of the function of the

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liver, the thyroid gland or other structures. On the other hand, intoxications having their origin within the body proper may be followed by a secondary intestinal intoxication; as in melancholia, in which in consequence of the diminished innervation and changed secretions, toxic materials may form in the intestinal tract.

From whatever aspect the subject is approached the problem presented is that of an intoxication in which disturbances of metabolism play the essential rôle. It is the nature of the intoxication which first claims our attention. The long duration of mental diseases points clearly to the fact that these poisons cannot be of a character such as to permit of elimination, at least not of ready elimination. Bouchard taught that the organism is a laboratory of poisons and this as much in the normal as in the pathologic condition. In the normal condition these poisons are in one way or another made harmless, but in diseased states, when the protective measures of the organism are weakened, these poisons were supposed to circulate in the blood and to be excreted by the urine. Bouchard, it will be remembered, worked out a method of determining the degree, that is, the coefficient of the toxicity of the urine, a method which later found application also in the study of the toxicity of the serum. Our knowledge of physiologic and chemical processes, however, was not sufficiently advanced in his day to permit of a correct interpretation of the results. The profound interest which these studies aroused was equaled only by the disappointment which in due course and necessarily followed. We all remember the extensive rôle that was at one time assigned to the uric acid group, a rôle that still demands consideration. It was at first believed that the presence of leukomains—as they were termed—in the urine was sufficient to account for the symptoms of a given case. All that it seemed necessary to do was to further the elimina-

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tion of these poisons, or to prevent their formation, or in some way to render them harmless; but, as we have seen, these expectations were doomed to disappointment. This was also true of the more elaborate studies made of the urine. The amount of nitrogen, sulphur and phosphorus excreted, the mineral waste and the output of water were all facts of interest; but they dealt largely with the end-results of metabolism and were obviously secondary to various recondite changes within the body.

An increasing knowledge of insanity has gradually indicated another view as to the nature of the toxicity in insanity. The clinical histories of many mental diseases—for example, of melancholia—consist of a period of gradual invasion, another period of maximum onset of symptoms and a third period of gradual subsidence. These facts suggest that we have to do with biochemical processes akin to those of infection and immunity, processes fermentative or enzymotic in their nature. In the prolonged intoxications encountered in mental disease the organism doubtless has recourse to the formation of immune bodies; that is, the organism reacts to intoxications just as it does to infections, namely, by a defensive fermentation. The rôle that the fluids of the organism play in its protection we have been taught by Buechner, Bordet, Bouchard, Behring, Roux, Kitasato, Ehrlich and others. The agencies at work have been shown to be substances which bring about immunity by a biochemical action, an action that is probably reducible to physicochemical terms. In combating the poisons which give rise to nervous and mental disorders, the phagocytes have, it would seem, no opportunity for the exercise of their special function, but it is not improbable that they join the serum of the blood in furnishing protective substances. The truths revealed in the discovery by Behring and Roux in regard to the curative and preventive action of the serum of

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animals immunized to diphtheria, in the discovery by Kitasato and Behring of the kindred facts in regard to tetanus and by Simon Flexner in regard to cerebro-spinal meningitis, constitute stories of brilliant human achievement. Ehrlich's results in regard to ricin and abrin and Calmette's in regard to cobra poison are likewise familiar to us all. Nor need we call attention to the specific serums developed against definite micro-organisms, the bacteriolytic serums or to the serums developed as defensive agents to erythrocytes, the hemolytic serums, or to the numerous other cytolytic and cytotoxic serums that have been experimentally produced. The doctrine of antigens and antibodies is destined, it would appear, to have an ever-widening application. An antigen is, as we all know, a substance which when introduced into the organism brings about the formation of a specific antibody. Among such substances are micro-organisms, their toxins, cells of various kinds or extracts of their substances, poisonous and nonpoisonous proteins and lipoid substances. Obviously, other facts, such as those of precipitation, agglutination, opsonin reaction and complement binding, must, like those of antigens and antibodies, be expressive of fermentation. Probably there is a common biochemical or physicochemical action basic to all of these phenomena. Certainly all of the processes of nutrition, all of the changes exhibited by proteins and carbohydrates, are the expression of such action and occur under the influence of catalytic and enzymotic processes. Again, the fact that ferments consist of two basic substances, first, a zymogen and, secondly, an activator, goes far to prove the identity of fermentation and immunizing processes. Again, just as antigens lead to the formation of antibodies, so do ferments lead to the formation of antiferments, and, finally, ferments likewise manifest the property of specificity, that is, each ferment is active on a specific substrate.

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The aspect of the subject of fermentation developed by Abderhalden depends on the fact that a foreign protein substance introduced into the blood excites the production of a ferment the action of which is to protect the organism against the foreign substance. Such a foreign substance may gain access to the blood from without or from organs and tissues within the body. An instance is the digestion of placental elements by ferments in the blood of the pregnant woman, the blood of nonpregnant women and of men containing no such ferments. Abderhalden and his pupils have already done a very large amount of work on this subject but we must content ourselves with the statement that Fauser and others have tested the digestive or reducing action of the serums of different forms of insanity in relation to the thyroid gland, the sex glands, the brain, the kidneys and other organs. Many remarkable observations have been made.

Three general statements in regard to the processes of fermentation appear to be justified: First, proteins and lipoids alone can evoke the formation of antibodies; in this process lipoids appear to play the rôle of activators, and their injection seems to bring about an increased formation of immune bodies. Second, the specificity of immunity and fermentation processes does not appear to be entirely absolute. It appears that the curative action of a given serum depends on its containing a larger amount of substances already normally present in the body. Third, in the struggle with intoxication and autointoxication, a most important rôle must be assigned to the glands of internal secretion. The various other tissues and organs of the body, in addition to their special functions, possess also the function of internal secretion inasmuch as they elaborate, as a result of their functional activity, and pass into the blood and other liquids of the body, certain substances which in one way or another act on other organs and thus add to the number of the

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hormones as we now term them. The life of the organism is that of a well-coordinated chemical whole. It is the disturbance of this coordinated chemical whole either by the introduction of foreign substances from without, or by the breeding of foreign substances within the body through the breaking down of the coordination, that causes intoxication, and it becomes clear that disturbances of metabolism, using this term in its widest sense, are the basic factors in mental diseases. How such disturbances of metabolism arise we have already indicated in the instance of the infections and poisons introduced from without. If the organism be essentially defective, deviate and aberrant insanity may of course arise without the influence of any external agency. The peculiarities presented by the defective organism are innate and often hereditary. Further they do not commonly manifest themselves in a marked degree until the period of life is reached in which metabolism assumes a special activity, namely, puberty or later. At this time, the sexual glands and with them all other glands of internal secretion and hormone producing structures and tissues take on a new rôle, enter on a new chemistry, and if the organism be badly constructed this chemistry becomes aberrant and toxic; for instance, in dementia praecox the sex glands appear to furnish materials directly toxic to the organism—that is, materials which act as foreign bodies and assume the rôle of antigens—while the defensive ferments, the antibodies, produced are in turn destructive in their action on the nervous system. In the manic-depressive group we have to do apparently with a somewhat simpler interplay of antigens and antibodies, but time forbids a further consideration of this interesting subject. Suffice it to say that in this group in which the picture is one of the predominance of antigens in the early stage—in an attack of melancholia or mania as the case may be—and of the predominance of anti-

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bodies in the later stage, it is by no means impossible that serums will be devised which will enable us to cut short such attacks and perhaps to practice preventive immunization for recurrences. It is with regard to the heboid-paranoid group, the great group of dementia praecox and paranoia, that the outlook is the most discouraging. Even here, however, it is not impossible that a fuller understanding of the problem will finally enable us to master the field; but if this is ever to be accomplished, it is only, I am firmly convinced, by further discoveries in the field which I have here so imperfectly outlined.

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OBJECTIVE PSYCHOLOGY OR PSYCHOBIOLOGY WITH SUBORDINATION OF THE MEDICALLY USELESS CONTRAST OF MENTAL AND PHYSICAL

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A few years ago the American Psychological Association discussed the question of a course in psychology for medical students. Since then the problem has been taken up in the Johns Hopkins Medical Department, last year in a course which I gave with the collaboration of Prof. J. B. Watson and Prof. Knight Dunlap, and this year in a course which I gave alone. The time allotted was two afternoon hours a week in the spring trimester of the second year, altogether twenty hours.

My first step was to find the standpoint of my students, many of whom had had courses in psychology at college. I asked them to state what relation psychology had in their minds to physiology and pathology. I was at once confronted with a lack of definiteness and agreement which is the inevitable consequence of the fact that there is but little mutual understanding among the teachers of psychology, and little applicability of what is taught either to the plain man's needs in viewing mental life, or to the physician's needs.

I found that most students had the conception that psychology deals with the mind as physiology deals with the body. They contrasted mental functions and physical functions, and had more or less vague notions of the doctrine of psychophysical parallelism which

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has governed science during the last fifty years or more. Most students, however, assumed an influence of mind on body and of body on mind after the fashion prevailing before the ingenious truce attained by Fechner's parallelism; and in this they evidently were prompted by plain sense rather than by any dogmatic theory. In the discussion there certainly was no evidence of any solid metaphysics worthy of that name and no realization of the often discussed and specially dreaded problem of interactionism.

I am not surprised at this state of affairs, for when I look around among my colleagues I find few well-defined attitudes. There are those who see no sense in studying mind beyond what is done in the physiology of the brain. There are, however, others who recognize the facts of suggestion, the rôle played by the emotions not only in hysteria, but even in Graves' disease; but just how the thing works, many believe to be beyond human ken. One of my colleagues expressed his views in last year's address before the Medico-Psychological Association, in which he emphasized the subjective character of the mental facts, suggesting in fact if not in word that mind can be of use to the physician in the form of symptoms, but not as causes and objective facts like the other objective facts of medical observation.

The difficulty lies in the hesitancy to accept a frankly biologic view of the reactions and behavior of man. As soon as mental attitudes and mental activities are accepted as definite functions of a living organism, mentation and behavior is treated as a real chapter of the natural history of man and animal, and psychology ceases to be a puzzle supposedly resisting the objective methods of science. "Mental attitudes" and "mental activities" certainly are doings and activities of definite individuals; we see them and prove them to be present or absent, or changed as attitudes and actions of others as truly as we know them in our-

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selves. We may know some of our own mental states in more detail; but that which counts is attitude, activity, and behavior, observable in any one, and sometimes rendered more accessible by inducing the subject to amplify his activities verbally or in other ways until we know the facts, that is, until they have been made objectively evident.

I am specially anxious to make plain what is meant by the fundamental assertion that what is of importance to us is as observable and as objective as any other fact of natural history. What is of importance to us is the activity and behavior of the total organism or individual as opposed to the activity of single detachable organs. It is more than cerebration; we must take our domain broadly as behavior and passive and constructive adaptation of the entire individual. It differs from the ordinary physiology because it represents an integration of biologic activity on a specific level through its having the characteristic of more or less consciousness and because of its hanging together by associative function. From the point of view of science, behavior and mental activity, even in its implicit or more subjective forms, is not more subjective than the activity of the stomach or the heart or blood serum or cerebrospinal fluid or the knee jerk. Each individual has his own mental activity, but to say that we cannot see it and make it accessible and understand it in others is a philosopher's scare like the statement that we can never know whether the world exists, because we know only mental states or processes. Common sense has never worried about the reality of the world. I hope we shall soon be agreed on the fact that we need not worry about the psychobiologic reality and the objectivity of those actions and internal workings of living beings which we call mentation and behavior. I should quit being a physician and a teacher if I felt compelled to doubt the possibility of my studying and knowing your minds

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and those of my patients well enough to draw practical conclusions from such knowledge. If solipsistic philosophy were true one might as well retire into absolute solitude. By making of mind something like the religious-philosophic concept of the soul, something opposed to the body instead of a function of the individual as a whole, traditional philosophy and psychology have rendered us a poor service.

The first step in a course of psychology for medical students is to restore in them the courage of common sense. It is not necessary to start the course with a discussion of the unconscious and the subconscious, and with hypnotism and psychanalysis and other specialized and more or less unfamiliar domains. On the contrary, I urge the student to trace the plain life history of a person and to record it on what I call the life chart; the result is a record of a smooth or broken life curve of each one of the main organs and functions, and in addition, a record of the main events of the life of the whole bundle of organs, that is, "the individual as a whole" and of the facts which determined and constituted his behavior. This realm of objective and determinable facts of the individual as a person constitutes what we as physicians need to know as psychology. The science dealing with these facts I call psychobiology, in order not to step on the toes of the introspectionist who might want to reserve the term "psychology" for the traditional type of subjective psychology. Its facts are behavior in the widest sense of reactive and constructive adaptation of the completely integrated organism. We ask: What are the individual's assets: the reactive and associative resources in the form of effective and expressive activity and its abortive forms, conations, affections, cognition, discrimination, and reconstructive and constructive imagination? Under what conditions are they apt to go wrong and under what conditions can they be modified again for the better? You can readily

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see that we are dealing with the absolutely objective and positive facts, peculiar only in the way in which they hang together by association in the wealth of equivalents and combinations, and in the varying extent and depth to which they implicate the parts of the integrated organism. So much for the student's general orientation with its restoration of the common-sense attitude.

As an instance of the study of assets, we take up the Binet-Simon and other genetic standards and survey the successive epochs of human life and their psychobiologic problems: infancy; then the period of acquisition of signs and language; the early childhood passing into what Joseph Lee calls the "Big Injun" stage and the school childhood; the preadolescent and adolescent period; the period of emancipation; the period of adult aggressive life; the period of maturity; the matron's period; and the period of senescence—each with its own psychobiologic features and problems.

Within this broad and clearly biologic frame, the student becomes ready to see a proper setting for the more detailed and specific chapters, among which I take up first the cognitive-representative data: reactions to things present (sensation and perception), reactions to things absent (memories and images), reactions with ideas and words and concepts or orientation; all activities depending more or less directly on the collaboration of brain and sense-mechanism. Then we take up the affective processes which determine the general trend of association and involve, beside the brain, mainly the sympathetic system and internal secretions, as Cannon has so well shown lately; and then the overt actions, specified as effective and expressive, and the laws of habit-formation, memory and association, and conation or will and its relation to instinct. We then consider the various degrees of consciousness and attention; and we introduce the data

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of hypnotism and the subconscious determining influences shown in the association experiment.

On this ground, we take up the genetic account of several leading functions or instincts as given in Pyle's "Outlines of Educational Psychology" and incidentally the psychobiology of sex life.

A review of the nonmental components of the mental integrations, the foundation of fatigue, and waking and sleep states, of the share of the circulation, of internal secretion, and of brain organization, rounds off the course and leaves us with a well-checked outline of examination of the mental resources and reactive tendencies of any patient, to be used and developed in the third and fourth year courses in psychopathology and psychiatry.

Psychobiology as thus conceived forms clearly and simply the missing chapter of ordinary physiology and pathology, the chapter dealing with functions of the total person and not merely of detachable parts. It is a topic representing a special level of biologic integration, a new level of simple units having in common the fact of blending in consciousness, integrating our organism into simple or complex adaptive and constructive reactions of overt and implicit behavior. I contrast (1) mentally or more or less consciously connected reactions, and (2) nonmental reactions of individual detachable organs which may as well be studied in the test tube and isolated and then certainly give us no evidence of consciousness. With this frank contrast we avoid panpsychism and solipsism and absolute subjectivism and all the other bugbears confusing the medical and lay mind and the would-be scientific psychologists. We keep on the ground of common sense and teach the student to study individuals or persons, in addition to making a study of the special organs, and in this study of individuals we deal definitely with objective facts which we must learn to specify and demonstrate, to account for genetically, to study for their effects and

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for means of modifying them for better or worse, just as we study the heart functions and not only the murmurs and the lesions, and the kidney function and the carbohydrate regulation or any other objective fact of biology and physiology and pathology. Reduction of the essential facts to terms of an experiment of nature, and study of the modifiability of the experiment is the fundamental law and aim of medical science, and this holds clearly enough for the psychobiologic assets as well as for any other type of reaction.

In harmony with this conception, the concepts of mental and physical must undergo a readjustment, as the concepts of sunrise and sunset had to undergo a reinterpretation. Science deals with a world of things, facts, and relations appearing in several distinct levels or types of integration. Physics deals with one set of aspects of matter and ether; chemistry with another, namely, the laws of behavior of atoms and their affinities and combinations; physiology with a biologic level, that is, those objects and their parts which grow by reproduction and metabolism; and as psychobiology we treat the functions of total organisms which blend in more or less consciousness in a manner constituting a special level of integration which has been especially and most characteristically enriched by the interindividual and social development of language. This level of integration we treat as psychobiology when considered as actual functioning and behavior of living organisms. All that which constitutes psychobiology to the physician is, therefore, also physical as well as mental. We can further recognize an ultrabiologic level of facts when we consider the products of such functioning, as logic and mathematics or theory of relations, or as history and record of the human race, including also the more than biologic realms of fact, philosophy, and religion. In this way we obtain an orderly perspective of the various sciences, but eliminate the contrast between physical and mental.

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It is desirable, I think, to make the student feel that he does not have to draw too sharp a line between mentally integrated and nonmentally integrated activities. Many reflexes or instincts or reactions can appear on the physiologic level or on the psychobiologic level. The difference lies in the mode of hanging together, the setting and the extent and kind of possible interrelations.

On the physiologic level the reactions last as long as the stimulus, are commensurate with the stimulus, and occur as it were according to the laws and requirements of contiguity.

The entering into the psychobiologic level brings in a more extensive scope of potential links and interrelations with the laws of consciousness and of associative relations.

The reaction becomes part of what I describe to the student as a burst or geyser of daily activity, with laws of blending and laws of falling into trends laid down according to the principles which experience with this special level of biologic regulations must furnish us. The student must realize that the mechanisms of the psychobiologic level are not limited to the type of consciousness which we know in waking life and which most psychologists cultivate exclusively. Watson's work shows that there are many problems which can be studied accurately with practical disregard of the "contents of consciousness," as we are apt to call the "implicit" links of action. Many conditions will, however, become intelligible only if we take into consideration the special characteristics of special types of consciousness, such as the dream states, half-dream states, states of distraction, hypnosis, and special affective states, the study of which can do full justice to the fact that some of these special states will prove open to explanation and reproduction or at least facilitation by the introduction of detachable physiologic lifts, chemicals, narcotics or internal secretions, of

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which we know that they can produce modifications of the hanging together of psychobiologic trends.

To sum up: From time immemorial the physician has been urged to treat not only the diseased organs or the condition, but also the patient. And of late years we have learned to realize that indeed some disorders can be explained, and treated, as abnormal and unhealthy ways of the person rather than as disorders of any one special organ; rather as disorders of the combination and its behavior. Every medical teacher gives some advice on this topic of doing justice to the personality; oftenest, and probably too often, he feels that the talent for dealing with personality must be inborn and cannot be hammered in by any training.

Of late years the difficulties in the total unified adjustment and behavior of our patients have been more and more reduced to intelligible processes: defects of development, intellectual, affective, or conative, improper use of the assets, etc. Instead of mere vague ideas of something requiring special handling, we have today a growing body of facts in the way of habit disorders, harmful substitutions and the like which must be heeded in helping the patient to reach his best ground. How can the student be helped to command these facts most quickly? We teach him physics and chemistry and biology and physiology. Who teaches him the elements of these reactions which we call behavior and mentation? He enters on this field with confused and confusing notions; how is he to straighten them out?

The first condition for productive work in this field of psychobiology as in any other, certainly, is controlled procedure and methods of description and record and experimentation which come up to definite standards. It is clear that psychobiology and psychopathology have principles to offer which go beyond the dilettante realm and form a fairly clean-cut body of methods and helpful procedures: not only safe neurologic methods and safer methods of work with internal

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secretions as such, but also safer methods of inducing and directing associative and nonassociative processes and associative material serving the best demands of the various types of make-up.

The difference between modern psychology and the older form is that we can no longer be satisfied with mere plausible statements and amplifications of the obvious, but must test and verify objective facts under controlled conditions and controlled modifications.

In my own teaching, I am determined not to put curiosities and the sensational to the forefront. Students whose interest goes toward such sensational things can in time be reached even with this conservative scheme if they have the right stuff in them. On the other hand, stagy psychology can be maintained to advantage by but few teachers and few workers and it fails with the bulk of our clinical method. We must discourage the ever-lurking interest in the occult and semioccult and replace it by solid confidence in reliable methods and by determined interest in matters obviously calling for serious objective study.

This hasty survey might easily gain by further amplification. I trust it may give an idea of the entire practicability of a course of study of objective behavior and mentation useful and essential to every student and physician; likely to give a sane balance and to furnish protection against extreme and one-sided vagaries or equally unnecessary psychophobia, which have so long marred the efforts of physicians in their working with the personality of the patient as well as with the individual organs. Saner views will make us physicians juster and more capable and less likely to drive a lot of sensible but misguided people into obscure mystical cults and ourselves into passing fads and fashions, and empty squabbling before a justly distrustful public, and they also will make unnecessary that new profession of "clinical psychologist" and efficiency psychologist for which a medical training will

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become obligatory if we physicians show the sense and capacity to furnish the needed psychobiology as a necessary and obligatory part of our training.

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ABSTRACT OF DISCUSSION

DR. THOMAS J. ORBISON, Los Angeles: This matter of parallelism I have not been able to grasp. Consider the phylogeny of the physical part of us and of the mental part of us—I cannot see when they join. Take our nervous system, from the ovum down through the nine months of intrauterine life in which evolution has speeded through so many centuries. We know that in a moment the organism is ready to go from one element to another, and the first breath the infant receives has changed that child so that it can never return one step. I have never been able to determine to my own satisfaction (and I suppose I never shall) how the physical and mental interact; how really intimate these two are, and where they differ. Probably I do not make myself clear, yet I would like to know more about what parallelism really means—how the mental and the physical come together, and is the soul a negligible quantity? If Dr. Adolf Meyer can understand such an ambiguous question as that, I would like to hear more explanation.

DR. JOSEPHINE A. JACKSON, Pasadena: I was glad Dr. Meyer brought up the point that personality is not something we are born with, but something we can have developed in us—something that can be hammered into us. Possession of "personality" connotes a knowledge and understanding of the reactions of the individual as determining his behavior. Each individual, because he is an individual, is different from everyone else; each has to be handled for himself alone. As Dr. Meyer said, treat the patient, not the disease. The individual differs in different periods of his life in his reactions; certain accidents and environments make him different; if we study we can understand and it will help us to handle the patient.

DR. ANDREW L. SKOOG, Kansas City, Mo.: I liked very much the statement Dr. Meyer made regarding common sense. He has gone over the field of preparation of the medical student, before his course of study, in psychology or morbid psychology, and the more or less confusion which exists in his mind. I think when we are teaching students either neurology or psychiatry, we should constantly bear in mind common sense. We are dealing with a complicated subject, perhaps complicated because of the preparation of the student when he appears before us. Therefore, to reiterate, bringing before the student common sense ideas is of the greatest importance.

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The subject of objective psychology has been discussed thoroughly. I admire the work of Bechterew which has been done with animals. The study of animals is very interesting, and to these investigations may be added also that of infants and young children.

DR. CECIL E. REYNOLDS, Los Angeles: I would like to mention one point in connection with the question that Dr. Orbison has brought up. If we assume, accepting Dr. Stoddart's views, that disturbance at the synapses is a cause of confusional insanity—that the normal relation of the dendrites of the cortical cells is disturbed—then we can bring into relation the views that Dr. Dercum has expressed on the toxic origin of mental disease with the psychical manifestations we are familiar with; for example, in confusional insanity. In the same way, if you regard the condition of sleep as a normal retraction of the dendrites, it is conceivable that those things that most affect our mind in the waking state will cause persistent interlacing of the dendrites concerned, with the resultant persistence of thought, especially deeply rooted desires, symbolized and distorted in a dream.

DR. ADOLPH MEYER, Baltimore: It would be unfair to delay the work of the section by an attempt to answer in full the question of Dr. Orbison, because it would mean to reiterate a great deal of what is contained in the paper. I can best illustrate what I mean by referring to the remark of Dr. Reynolds. He has drawn our attention to Dr. Stoddart's view of confusional insanity, as if he thought all mental disorders should be explained by neurone-retraction. Dr. Stoddart has, by the way, lately come out as the most extreme Freudian in the English language. I would like to ask who on earth ever saw those dendrites wiggle in exhaustion psychoses. How much more important it is to look for what we can see. We want to rehabilitate the study of that which we see in every patient; to study every activity, every function of the individual or personality as a whole for all it can teach us, before we enter on confusing discussions of pure and often improbable hypotheses. I eliminate psychophysical parallelism because it brings more confusion than help. To my mind there does not seem to be any necessity of cultivating confusion over that which does not exist. I think it is greatly to the advantage of our science and our standing before a sensible public if we speak mainly of things which we can demonstrate, rather than to speak about synapses, hypothetical disorders, of which at the present time very few people know anything. The main purpose of the introduction of an objective psychobiology into the range of objective facts of physiology and pathology is to enable us to remain on a ground of common-sense and direct observation of the activities of the individual as a whole—facts which we cannot afford to ignore or to handle without training.

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DELUSIONS OF PERSECUTION AS A BIOLOGIC DEFENSE REACTION

CHARLES LEWIS ALLEN, M.D.
LOS ANGELES

The life of the individual is a constant struggle, and is made up of a series of reactions having as their end the opposing of influences which tend to limit this life, or at least to prevent its development along the most favorable lines. Psychologists have come to believe that these reactions, which in their sum go to make up human conduct, are determined, in the main, by inherited tendencies which have been evolved in the struggle for existence of a long line of ancestors dating from the lowest forms. These tendencies have been developed in order to carry out the three great ends of organized beings; namely, defense from danger, procuring of food, and reproduction. The mechanisms to accomplish the first of these ends, requiring the most prompt and active reaction — protective mechanisms — were earliest and most firmly established.

These protective mechanisms vary immensely, from the methods of concealment of the lower forms of animals, through the armor of the turtle and armadillo, the venom of the serpent, the offensive odor of the skunk, the fleetness of the hare and the antelope, the teeth, the claws, and the strength of the tiger, to the capacity of man through his superior knowledge and reasoning power to invent weapons which more than compensate his lack of speed and strength.

Not the least of these human defense mechanisms, however, is developed in the ability of man to analyze the conduct of his fellows and to provide against any manifestations of hostility on their part. This ability

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varies immensely in different individuals, depending on their mental makeup, education and environment, state of health, good or ill fortune, etc., and in accordance with the laws of adaptation it tends to be sharpened by adversity and dulled by ease and comfort. Also, for the development of the critical faculty, we are largely dependent on the harmonious working of the somatic and the psychic functions. The deformed or sickly individual is only too apt to be exaggeratedly sensitive and, expecting slights and opposition, to react excessively to trivial causes, and a similar attitude is even more frequently dependent on defective mental makeup. From the other side, an actually unfriendly attitude sharpens the critical faculty, and, in a person of morose and suspicious temperament, the reaction may assume a pathologic intensity. These instinctive defensive reactions, being earliest and most strongly developed, are apt to persist longest, and, in mental disorganization unchecked by normal inhibition, may be set off by stimuli ordinarily inadequate.

Our study of the somatic symptoms having failed to enlighten us greatly with regard to the phenomena observed in insanity, the tendency of late has been to return to the mental symptoms and to investigate them in the light of the reaction of the individual to influences from within and from without. To have pointed out this way is not the least service of the psychoanalytic school.

Certainly, among the most striking of all the symptoms of mental alienation is the development of delusional ideas. Among these ideas, those of persecution are especially frequent, and while they are particularly characteristic of paranoia, they are present, at times, in practically all forms of insanity.

Different as may be the modes of life of different peoples, ideas of persecution present, the world over, certain general similarities which alone would seem to indicate that they have some common basis.

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In common with other delusions, they center about the personality, the ego, of the subject, and have, as their content, the notion that influences inimical to him are at work, to the prejudice of his person, his rights or his honor, or are endeavoring to injure him through those dear to him. The means supposedly employed to this end have varied greatly with the state of culture and environment, the sorcerer and the demon having today largely given place to the forces of nature as utilized in electric apparatus, wireless telegraphy, the dictograph, etc., though the rôle of poison is still as important as ever.

The mechanism of delusion formation has been much disputed, but all authorities are agreed on the importance, in the genesis of delusions, of fallacious perceptions, and particularly of the affective state of the subject. Into the causation of hallucinations we cannot enter here. It is, however, common experience that actual perceptions are greatly modified by the emotional condition of the individual at the time and may be wrongly interpreted if this state varies greatly from the normal. Conceptions arising on a basis of actual, though falsely interpreted, perceptions are themselves falsified and are readily connected into a delusional system. Since delusions are not the product of previous experience, they cannot be corrected by reference to it, but they are doubtless modified by the strong hereditary tendencies already alluded to. For their development and formation, a total alteration of consciousness is necessary, so delusion formation indicates a "general disturbance of the total psychic condition . . . Delusions are aroused by the variation of the affective condition which translates slumbering hopes and fears into conceptions. That these conceptions become delusions is due to the failure of judgment, this in some cases, through pathological emotional excitement, in others through clouding of consciousness, in still others through a weakness of the

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understanding." (Kraepelin). Now all of our reactions are originally determined by sensations arising from within and from without.

Crile has developed this idea in his recent works on "Anoci-Association" and "The Emotions." He asserts that trauma to the chest and abdomen stands first in the production of shock, and suggests that this is because these regions are the most abundantly supplied with "nociceptors," probably since they were originally the special points of attack of beasts of prey; hence stimulation here calls forth the most energetic neuro-muscular defense reaction, and results in the greatest nervous exhaustion, which is translated as shock. This reactive tendency, transmitted through countless ancestors, has become so fixed that it persists today though the necessity for it no longer exists.

For Crile, the emotion of fear is the "representation of injury" and calls forth the same defensive mechanisms of muscular tension, increased adrenal and thyroid secretion, etc. These may be excited not only by actual physical contact, but also through the distant receptors, as by sight, hearing and smell.

Darwin long ago called attention to the fact that men through countless generations have attempted to escape from their enemies by fight, or by flight, and that the great exertions necessary in either case have, as their final result, palpitation of the heart, sweating, pallor, trembling and the prostration of complete exhaustion, phenomena identical with those of fear, and suggested that when fear is strongly felt, though there is no exertion, the same results tend to appear, through the influence of inheritance and association.

Cannon has shown that emotional states, such as fear, are accompanied by greatly increased adrenal secretion, which has the function of raising the blood pressure and energizing the muscles, while Crile's examination of the brain cells in animals subjected to fear alone, without trauma, has disclosed changes similar to those found after exhaustion and trauma.

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According to the last-named author, an animal "driven strongly by emotion" shows (1) mobilization of the energy-giving compound in the brain cells; (2) increase of epinephrin, thyroid secretion and glycogen, increased power of oxidation in the muscles; (3) accelerated circulation and respiration and raised temperature; (4) altered metabolism. All these to increase the motor efficiency while other organs not contributing to this end have their function inhibited.

Phylogenetic influences are so strong that though an enemy is not attacked physically, these weapons are nevertheless made ready. The emotion of fear cannot but suggest the idea of danger.

In the majority of cases of insanity, the earliest symptoms are unrest, sleeplessness and often disturbing dreams. The patient may be aware of a certain alteration in his consciousness of self and often enough we hear the complaint that "things look strange," and "everything is changed." We have all experienced the feeling of apprehension and unrest which seems dependent on sensations apparently rising up from the abdominal cavity, and the emotions so aroused are certainly akin to fear. Now, fear, being the emotional condition associated with the perception of danger, would naturally give rise to defense reactions, and if the cause for fear is unclear, no enemy being in sight, this defense reaction will not proceed to the actual physical preparations for combat, but only to the awakening of conceptions of danger which, on account of disordered judgment-forming capacity, take definite form as ideas of persecution.

It may be objected that in paranoia, the form of insanity characterized *par excellence* by delusions of persecution, the genesis of these delusions is slow and gradual, and can hardly be connected with an emotional state. Also, fallacious perceptions, though frequently present, are usually developed later than the delusions. Now, the paranoiac possesses an originally defective

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mental constitution, in some ways resembling uncivilized man in his egotism and lack of altruism. He is naturally distrustful and suspicious, and, while his reasoning power may be good, he misinterprets ordinary happenings in the sense of his exaggerated ego and is ever ready to see opposition, if not danger, and to react to these misinterpretations, much as the savage does to the unfamiliar, by a defense reaction, that is, by ideas of persecution. Again, according to the views of Specht, paranoia has in its evolution the closest relations with manic depressive insanity, the ideas of grandeur developing in connection with the exaltation and increased feeling of self of the manic phase, those of persecution being similarly connected with the affective tone of the depressed phase.

Such are the considerations which suggest to me that delusions of persecutions should be looked on as a species of defense reaction traceable to the inherited instinct of self-protection.

From the work of Cannon and others it would appear that the internal secretions, especially those of the adrenal, the thyroid gland and the liver, play a part in the preparation for defense and are increased in strong emotions, notably in that of fear. May it not be possible that we have in this fact some explanation of the circumstances that delusional conditions, especially delusions of persecution, are so frequent at the times of presumably greatest change in the function of the ductless glands, namely, about puberty and the climacteric?

Possibly the further study of the functions of these glands, so much in the minds of the medical profession today, may throw a more definite light on this subject.

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MYOTONIA CONGENITA

W. A. JONES, M.D.
MINNEAPOLIS

The various types of muscle-disease suggest to the general reader either a juggling of names or a confusion of ideas, and usually both. The literature, too, on the subject illustrates a diversity of opinion; but, out of the collection of reported cases, one may safely classify and differentiate clinical types.

Myotonia congenita, or Thomsen's disease, was first carefully described and recognized as a type by J. Thomsen, a Danish physician of Kappeln, Silesia. Thomsen was a sufferer from the disease, and came from a family in which it had existed for five generations, although the disease was described—at least a disease similar to myotonia congenita—by Charles Bell in 1832, but was not put in its proper category. Benedikt described a similar condition in 1864; von Leyden, in 1866; and in the same year Erb collected twenty-eight cases. Thomsen's paper, however, did not come out until 1876, and then was entitled "Tonic Cramps in the Voluntary Muscles in Consequence of an Inherited Psychical Disposition." [1]

Since 1876 the number of cases reported has rapidly multiplied; hence the confusion in the differentiation of these muscle-diseases.

In order briefly to summarize what is usually found under myotonia congenita, the following case will, perhaps, give an outline, although it presents no new features:

Mrs. E. S., aged 41, married twenty-one years ago, and the mother of six children, four of them dying within the first five months of disorders of metabolism. Two children are

1. Osler: Modern Medicine, vi, 395.

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living and well. The parents of the patient are both living. The father is 62 years of age and in good health. The mother is 58, and very gross in appearance, probably suffering from obesity. She lives in a distant city, and consequently it is impossible to investigate her present condition; however, it is stated by the patient that the mother has suffered from a cramp-like attack in both hands for a great many years. This is the only history the patient has been able to give as to the possible heredity of her present trouble. A brother of the patient is living and well, and two sisters are living and in good health.

Mrs. S. has always been below par physically, and during childhood suffered from measles, mumps, chickenpox, and repeated attacks of tonsillitis. She has never had diphtheria or scarlet fever.

At 29 years of age she had an attack of smallpox.

Her menstrual life began at 14, and at 39 the menstrual periods became irregular and scanty in amount. She never sustained an injury of any sort, except at the age of 29. Following the attack of smallpox she was operated on for tubal pregnancy.

For the past twenty years she has had marked varicose veins, especially in the left leg; and these have caused her a great deal of inconvenience, and she has the usual stinging sensations which occur in a varicose condition.

The patient uses no drugs or alcohol, but has the tea habit, drinking from eight to twelve cups per day, and, in addition, two cups of coffee.

She has no evidence of a circulatory or a respiratory disease, and there is no active dyspnea, nor any palpitation. Her digestive apparatus is good, although her bowels are somewhat irregular and she complains of the usual distention of the abdomen. She is able to do her housework, although under some hardships; and for years she has attended to all her household duties and the care of her children.

The patient remembers that ever since she was a little child she has had trouble in moving her hands. She recalls that they would be in a definite position, and when she wanted to move them she found it impossible, for a few seconds, to make the required muscular effort.

During her young womanhood she had at times some difficulty in talking, and in other uses of her tongue. She says that her tongue would become spastic when she attempted to speak, or to move it in eating and swallowing. She described the sensation as a cramp in the tongue, similar to the cramp-like sensation in her hands. This condition has largely disappeared as regards the tongue; that is, she is able to speak clearly and distinctly, has no hesitancy in the movement of the tongue, nor has she any difficulty in swallowing. Occa-

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sionally, after shutting her jaws she finds it difficult to open them; the jaw feels as if it had been locked. These attacks, or tonic spasms of the jaw, never last more than a moment.

Six years ago, during her last pregnancy, when she was 36 years of age, the patient noticed that there was some difficulty in her feet and legs. The onset was very gradual; but it slowly and progressively grew worse. At the present time, if the patient attempts to go upstairs, she places one foot on the step, then the muscles become locked, and it is impossible for her to move her other foot and leg for several seconds. She also has difficulty in getting on the streetcar. With one foot on the lower step and the other foot either on the ground or in midair, she cannot get into the car until the muscles unlock. She also has difficulty in getting up from her chair. Various attempts are followed by a spasm of the muscles, but she is finally able to arise.

When the patient is lying down, and has no weight on her feet and legs, she is able to move them in whichever direction she pleases, and the movements of the hands and arms also improve when she is lying down, although, occasionally, she has a spasm, and it is more or less difficult. Repeated efforts on her part, either to move her arms or legs, render each movement easier.

When the muscles are stimulated by electricity, either the faradic or the galvanic current, they promptly respond to the stimulation, but the relaxing movements require from four to eight seconds. The muscles of the forearm and the arm, as well as those of the leg, show this same electrical reaction, and the same hesitancy in their relaxing movements. This applies particularly to the movements of extension. There is some spasticity on passive movement; and her gait is more or less spastic until she is thoroughly under way. She has no contractions, and no muscular atrophy, except that the left lower face exhibits a little restriction of muscle movement, that is, it does not respond as promptly as the muscles of the right side. The movements of the upper face are plainly present, but weak. She has no atrophy of other face muscles, or of the sternomastoid or the vasti of the thighs.

The reflexes are obtained with some difficulty. They are feebly present in the biceps, are obtainable in the triceps, and are absent in the Achilles on both sides. The deep reflexes of the patellar group are obtained only on reinforcement. There is no clonus. She has flexion from plantar irritation, but there is no Babinski or other phenomena. Her grasp is rather feeble, as shown by the dynamometer; and she is able to force the instrument only up to point 7.

Her general condition is poor; she is pale and stringy looking, and has a tired, woe-begone expression. She has a small goiter, which involves the right and middle lobes. It

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is soft and a little boggy, but there are no other symptoms, although this condition has been present for about eight years.

The teeth are poor, and she has a decided pyorrhea alveolaris.

The bladder control is a little impaired, but not to any degree which renders her at all uncomfortable, perhaps no more than the average woman who has had six children.

The pupils are equal in size, and react to light and accommodation. There is no disturbance of vision or of any of the other cranial nerves.

A section of the muscle was removed from the upper arms, from the triceps area, and the following microscopic findings obtained, the description being from sections of muscle mounted and stained with hematoxylin and eosin:

The tissue seems to stain somewhat deeper than normal muscle, and in places shows marked affinity for the hematoxylin, but no degenerative changes can be distinguished. No distinct vacuolation was noted, although in places it might appear as such; but in all probability, it was due to separation of fibrillae or to an artefact.

By far the most striking features to be noted are the increase in size of muscle fibers and the presence of numerous nuclei, arranged, as it would seem, in fairly definite order.

The increase in size of the muscle fibers is very marked, varying from two to three times the normal size of muscle fibers. A lack of normal parallelism, while present, is but slight, especially when the size of the fiber is considered. The various muscle bundles do not appear as compact and firm as normally, but seem more loosely bound together, the individual fibrillae being more or less separated. Crossed striation, which has been reported to be absent in some cases, is certainly but feebly marked in places, while in others it seems to be absent.

The abundance of nuclei which stain a deep blue, and apparently do not partake in the general increase in size, is of interest. Just why they exist in such numbers and arrangement is not easily explained. From longitudinal sections it would be difficult to say whether the nuclei were in the muscle fiber or about it. From cross-section it would seem, however, that the nuclei arrange themselves about the muscle fiber and in or beneath the sarcolemma. These nuclei are usually in single rows, of varying length, and not confined to any portion or section but very numerous and all fibers show presence of these rows, as a rule but one row to a fiber, but this is not constant, as in places there would seem to be several rows. This may be due to the overlying sarcolemma sheath, or adjacent muscle fiber. These nuclei vary from six to thirty in a row, placed end to end.

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A connective tissue change has been described; in the present case, except for a moderate looseness of separate muscle bundles, no change is to be noted.

The etiology of this case is rather obscure, except that it presents a very marked suggestion of heredity.

One cannot but consider that the exanthematous diseases that this patient went through in early childhood may have contributed to the early appearance of her symptoms. Then, too, she was a slight, frail, and rather poorly nourished type of woman. Also, the fact that four of her children died from nutritional disorders may tend to emphasize the theory of Dr. Ralph Pemberton,[2] who claims that in most of the cases of myotonia congenita there is a lessened creatinin output and a marked loss of calcium and nitrogen. This may or may not be an important factor, although in the majority of cases of myotonia congenita which come under observation, the patients are discovered to be suffering from the disease at about the twentieth year. Not uncommonly, however, children under the age of puberty are found to exhibit these awkward movements, which prove, under examination, to be a tonic spasm. It has been recorded by most observers that the majority of cases of myotonia congenita occur in men, and the lesser number in women. Just why, it is not easy to determine. The diagnosis of this case rests on the myotonic action of the muscles; that is, the response to stimulation and the slow relaxation which follows the voluntary contraction, the attitude of the patient, the character of the movements when an effort at standing and walking is made, and the use of the hands and arms are all a part of the myotonic action.

The prognosis in this case corresponds to other cases reported and depends on the severity or extent of the initial spasm. Not infrequently these people are able to carry on their work, as is the case with our patient.

2. Pemberton, Ralph: Jour. Med. Sc., cxli, 253.

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The disease, however, is essentially a chronic one, and progressive, with minor relapses or remissions.

The treatment is purely speculative. Various things have been done, but drugs have not proved advantageous; in fact, no permanent benefits have been realized from exercise, massage, or baths. The only thing that seems definite in the treatment of the disease is to keep the patient as active as possible; and, if there is a question of loss of lime salts, they can be introduced. These patients should, however, be kept from undue excitement or undue exposure to cold.

In connection with the report of this case it might not be out of place to refer to a somewhat similar condition which is known as Eulenberg's disease, or paramytonia congenita, an affection something like Thomsen's disease, in that the tonic spasm appears in certain voluntary muscles, more particularly in the face, so that the patient is unable, for fifteen minutes or longer, to open his eyes or to speak. The tonic spasm in Eulenberg's disease is usually caused by cold, but not by exertion as in Thomsen's disease.

Dysbasia angiosclerotica, or "intermittent limp," occasionally found in man, is found analogously in horses and is called "spring-halt." This condition is found in middle life and among gouty people or excessive smokers. On walking, the legs tire, and are heavy and painful with a cramp-like pain or limp. Persistent effort increases the difficulty, and it is associated with mottled, purple or red spots over the area of distribution of the posterior tibial or dorsalis pedis artery, due, supposedly, to a temporary spasm of the sclerotic vessel.

Tetany and myotonia congenita may be associated. Paolo Blau[3] records such a case. A young man of 21, a soldier, gave a history of symmetrical spasms of muscles of both extremities, the voluntary muscles particularly being involved. While marching he

3. Blau, Paolo: Wien. klin. Wchnschr., March 18, 1915.

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found it impossible to continue. The attacks came on after a long period of rest, and lasted from four to fifteen minutes. They began by a drawing pain in the neck, paresthesia, and a heaviness in the lower extremities. It was thought that these attacks were brought on by excitement. A sister and an uncle of the young man were similarly affected.

Myotonia congenita may also have associated with it von Graefe's sign.

Dr. J. P. Sedgwick[4] calls attention to von Graefe's sign in connection with several cases of myotonia congenita. He was able to collect histories of twenty-nine members of a family, thirteen of whom were affected with myotonia congenita, and sixteen were free. No atrophies were recorded, but all of the affected cases showed the von Graefe sign to a greater or less degree.

The most common muscle disorder with which Thomsen's disease may either be confused or associated is myotonia atrophica. This disorder or type is characterized by the rare association of muscular atrophy with slow relaxation of muscles after voluntary contraction. The weakness of the facial muscles, giving rise to the myopathic face, marked atrophy of the sternomastoids, the vasti of the thighs, and the dorsiflexors of the feet, are characteristic and salient symptoms. This name, "myotonia atrophica," was first suggested by Rossolimo, but others have described it as Thomsen's disease with muscular atrophy.[5]

Dr. Fred E. Batten,[6] in an article on "Myopathy," records a number of these cases and states that the condition of the muscles is the same as in other myopathic cases. He quotes Steinert as having found degeneration of the posterior columns in the lumbar cord, thus differentiating it from Thomsen's disease.

4. Sedgwick, J. P.: Am. Jour. Med. Sc., July, 1910, p. 80.
5. Batten, Fred E.: Brain, 1909, p. 87.
6. Batten, F. E., in Allbutt and Rolleston's System of Medicine, vii, 31.

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Dr. Batten believes that these two diseases—that is, Thomsen's disease and myotonia atrophica—are simply variations in type or that they belong to the mixed types in which there may be intermediate forms or symptoms which serve as connecting links between types. It is interesting to note in connection with the term "myopathy" that there are four or five groups which are described under this common heading, which do not belong to the myotonia congenita group unless we accept the statement of Batten that they are all varieties of the same type of disease. For instance, in simple atrophic myopathy there is a lack of power and tone in all the muscles of the body, and to these Batten has given the name of "infantile." This disease is congenital or starts in early infancy and advances but slowly; and in some instances the patient is able to develop certain groups of muscles. It exhibits none of the typical myotonic muscular actions. In the next group comes pseudohypertrophic paralysis, in which there is weakness of certain groups of muscles with hypertrophy of some and wasting of others.

This disease is quite commonly known, and hardly merits further description, except that it may be combined with myotonia. Arthur F. Hertz[7] records a family in which the father and mother are normal, but the oldest son, aged 25, and the oldest daughter, aged 18, and the youngest son, aged 13, were all affected. A fourth son, who was drowned at the age of 18, was "also very slow" in his muscular movements. The generalized muscular hypertrophy is illustrated in each case. All showed myotonic action on voluntary contraction. Just another case to add to the confusion.

One form of myopathy is the juvenile form of progressive muscular atrophy, which is characterized by a weakness and wasting of the muscles about the

7. Hertz, Arthur F.: Proc. Roy. Soc. Med., 1914, vii, 139.

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shoulder girdle. Erb first described it, and probably it is a type of the living skeleton whom we formerly saw in the museums and other exhibitions. In this type of muscular atrophy there is no myotonic action.

The next form is the facio-scapulo-humeral form of Landouzy and Dejerine, which involves the face muscles, the shoulder, the pelvic girdle, and the muscles of the upper arm and thigh. The weakness of the face muscles is often present at birth; and here, too, this disease has none of the symptoms which characterize myotonia congenita. Then, too, there is distal myopathy, described by Sir William Gowers in 1902, and also recorded by Spiller, Dejerine, Thomas, and Campbell. The salient features of this type are a weakness and atrophy of the muscles below the elbow and knee, while the proximal muscles remain well developed.

Family periodic paralysis is another peculiar condition which might be confused with myotonia or myopathy. The paralysis, however, which is the characteristic feature, is very unique. If the patient is seen during the attack, the diagnosis is very simple. The paralysis, which may last from hours to days, is unaccompanied by any disturbance of sensation or disorders of consciousness, is irregular in its periods of occurrence, and is followed each time by a complete recovery.

Perhaps I may be pardoned if I refer, briefly, to another disorder or disease of the muscles which is commonly known as Oppenheim's disease, and described by him as myatonia congenita and by others as amyotonia. It has also been termed congenital hypotonia, or a myoplasia, and is known and referred to by Batten as the "infantile type of myopathy."

I simply introduce this as an excuse to call attention to the similarity in names rather than to discuss the condition, which is quite unlike a myotonia congenita. All of these terms include a condition in

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which there is an extreme flaccidity of the muscles, most marked at birth or early childhood, and associated with entire loss of the deep reflexes, and great weakness, but no absolute paralysis of any muscle. The limbs are most affected, and the face is almost always exempt. The muscles are small and soft, but there is no local wasting. The limbs are so flexible that a child or infant may be lifted up by an arm or a leg; and the joints and muscles and trunk seem to hang down as if without any support whatever, and certainly without resistance. In these amyotonias contractures are prone to occur in time. The faradic excitability of the muscles is increased, and strong stimuli are borne without complaint. It is claimed there is a tendency to slow and progressive amelioration. Notwithstanding the difference in opinion as to the amyotonias and myopathies, they are evidently separable and distinct clinical types of disorder. In discussing the differences between these types of disease, Collier and Wilson[8] draw the following conclusions:

1. The myopathies are conspicuously family diseases, whereas no family tendency has been recorded in amyotonia.

2. Several types of myopathies often show family relationships, one with another, whereas no case of amyotonia has been recorded in a myopathic family.

3. A large majority of the cases of amyotonia are congenital, the conditions being obvious at birth.

4. In a minority of the cases of amyotonia it has appeared acutely, and has reached its most severe degree in a few days. In none of its several types is myopathy apparent at birth nor has it appeared acutely or reached its maximum in a few days.

5. Local muscular wasting, which is a marked feature of myopathy, is not present in amyotonia.

8. Collier and Wilson: Brain, May, 1908, p. 1.

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6. The course of myopathy is one of progressive increase in muscular weakness; that of amyotonia is one of progressive amelioration of the symptoms.

7. The return of the deep reflexes in amyotonia may occur after months or years, while in myopathies the deep reflexes remain absent.

8. So far no nerve trunk or spinal-cord lesions have been demonstrated in true amyotonia and it may be added that no nerve or cord lesions have been found in true myotonia congenita.

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ABSTRACT OF DISCUSSION

DR. FRANK B. STARKEY, Philadelphia: This subject of myotonia and myopathy has interested me very much because it has been attributed by a great many to disturbance of the glands of internal secretion. So far as I know, there is nothing definite to demonstrate this, and from the standpoint of treatment I do not believe that this theory can be borne out. I have had occasion to treat quite a large number of these cases, and they did not seem to respond to treatment along the lines that usually prove effective in cases of disturbance of the glands of internal secretion.

DR. G. W. ROBINSON, Kansas City, Mo.: In his closing statement, I believe, Dr. Jones said that no central nerve lesion had been found in true myotonia. Previously, I understood him to say that Batten had found degeneration of the dorsal cord tracts. One interesting feature is this: we cannot diagnose these cases (myopathies or myelopathies) without the clinical history. Wilson says that there is no differentiating pathology which will enable us to make a diagnosis by examination of the muscles. Many theories have been advanced, but it occurs to me that we should consider the antagonism between the cerebellum and the cerebrum in relation to muscle tone. The cerebral cortex inhibits muscle tone, while the cerebellum accelerates it. Whatever the condition may be, I believe there is some difficulty in the origin or discharge of nerve impulses in the control of the muscle tone, as we have a muscle that contracts and will not relax readily. We have in myotonia an accelerated tone which means the overaction of the cerebellum or underaction of the pyramidal mechanism.

DR. FRANCIS X. DERCUM, Philadelphia: I would like to ask Dr. Jones whether the affection described by Dr. Rich of Ogden, Utah, about twenty-one years ago is not identical with that of Ehrenberg?

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DR. W. A. JONES, Minneapolis: The reference which Dr. Dercum has just made to Dr. E. I. Rich's observations in Utah, I know nothing of, consequently I cannot adjust the dates as to whether Rich's observations antedated those of Dr. F. E. Batten. Dr. Robinson evidently misunderstood the paragraph in my paper. I, quoting from Batten, he quoting some one else, said that myotonia atrophica had been found to be associated with some degeneration of the spinal cord, consequently it was not a true myotonia congenita. Perhaps after all Dr. Thomsen was quite right when he said the disease myotonia congenita was one in which the psychical influence was the predominating factor.

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CHRONIC TETANY

REPORT OF CASE

J. P. MUNROE, M.D.
CHARLOTTE, N. C.

Clinicians are constantly on the alert to find a definite pathology for those conditions classed as functional diseases.

In recent years an unusual amount of attention has been directed to the so-called "regulators" of metabolism, namely, the glands of internal secretion, and the vegetative nervous system.

In my opinion, many cases, heretofore designated neuroses, on thorough investigation would be found to have a pathologic basis along the lines indicated. In this particular class of cases, the parathyroid gland is perhaps more often at fault than is generally supposed. Along with this, a serious want of balance in the vegetative nervous system is often an important factor.

The classical definition of tetany is: "It is a disease whose symptom complex consists essentially in intermittent bilateral muscular contractions which are painful in character, and usually without loss of consciousness."

The pathology of tetany, as understood today, is based on the postmortem findings, the results of operations and therapeutic tests.

It is generally agreed that the underlying factor is deficient parathyroid secretion, that this causes deficient calcium content in the blood, and that a hyperexcitability of nerve cells and fibers results from this calcium deficiency. There are those who believe that

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this irritation affects chiefly the central nervous system. MacCallum's experiments, however, indicate that it is peripheral nerve irritation. It is probable that both the nerve cells and the nerve fibers are irritated.

It is interesting to note that Sabbatini's experiments with trisodic nitrate, a salt of citric acid, which renders inert calcium salts, will if injected into the system, cause tetany.

I wish to state parenthetically that in the treatment of the case here reported, the fruits containing citric acid seemed decidedly harmful, and it was only after fruits of this kind were prohibited that any improvement was attained.

Berkley and Beebe advanced the theory that the most important factor is a large increase in ammonia, resulting from certain metabolic changes, endogenous toxins or from the introduction of exogenous toxins into the system. They have produced tetany by injecting ammonia, and then relieved it with calcium salts.

Operative tetany has occasionally occurred from the unintentional removal of the parathyroid glands in operating on the thyroid. Crocker reports five cases in 1,000 operations, and Frazier was able to collect eight cases in 2,000 operations.

The latest case reported that has come under my observation was reported by Johnston and Budd, at the Southern Medical Association, November, 1914. In this case, symptoms of tetany appeared thirty hours after operation, and death occurred in fifty-two hours, preceded by tetanic convulsions. The difficulty of protecting these little glands in thyroidectomy will be apparent by a brief reference to their anatomy. They consist of four small lobes of two on each side, each 6 to 7 mm. by 3 to 4 mm. by 1 to 3 mm. They lie behind the thyroid gland, and are closely adherent to it.

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It is well at this point to refer briefly to the autonomic nervous system. We have two separate and distinct autonomic systems, enervating, smooth muscular fibers and the secreting glands, namely, (1) the sympathetic system proper, and (2) the autonomic proper, also called the vagal system in the wider sense.

Both these systems appear to be acting constantly, and in a certain sense are antagonistic to each other. A certain tonus exists in each system. Under normal conditions there is a sort of balance maintained between the tonus of the sympathetic proper and the autonomic proper or vagal system. For example, in a dim light the pupil is kept half way between contraction and dilatation. If the third nerve of the vagal system is stimulated, contraction results, whereas if the sympathetic system, which supplies the radiating fibers, is stimulated, dilatation would result.

Similarly the heart's contractions are kept at about 72 per minute under the balanced action of the accelerator nerves of the sympathetic system and the inhibitory nerves of the vagal system.

Both systems seem to be under a double control: (1) control exerted through the cerebrospinal axis; and (2) control dependent on the direct chemical action on the autonomic nervous system by substances called "hormones," which are derived from the glands of internal secretion. Sympathetic tonus appears to be kept up by a constant flow of epinephrin.

The posterior lobe of the pituitary gland has a similar action, selective, especially on the uterine muscle.

We do not know of any internal secretion exerting a similar effect on the tonus of the vagal system.

There are medicines, however, that are known to increase this tonus, and some have a distinctly stimulating effect. Then again, there are medicines which have a paralyzing effect. These facts form the basis

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of certain therapeutic tests. The medicines commonly used for these tests are epinephrin, atropin and pilocarpin.

Epinephrin increases sympathetic tonus and thereby tends to balance excessive vagal tonus.

Pilocarpin stimulates the vagal system, at least parts of it, and thereby balances excessive sympathetic tonus.

Atropin tends to paralyze or depress the nerves of the vagal system, and thereby has practically the same effect as epinephrin in cases of excessive vagal tonus.

Kahn and Falta, after demonstrating that tetany is not confined to the voluntary muscles, but extends as well to the autonomic nervous system, produced some evidence indicating that tetany is essentially a disease of excessive vagotonic condition of the nervous system. The case which I report seems to lend support to this view. Falta, by using a dose of pilocarpin that has practically no effect on a normal person, has shown that the same dose may produce acute dilatation of the stomach and cessation of peristalsis, if given in a case of tetany.

This suggests an interesting question, whether the term gastric tetany should be used to designate a distinct type. Jelliffe and White (page 109) say, "The course of tetany in gastric dilatation is toward a fatal termination, unless there is surgical intervention or relief of the dilatation." Cases of gastric tetany reported (Osler) are about thirty, with a fatality of 33 per cent. for patients operated on and 67 per cent. for those not operated on.

This would seem to indicate need of operation in cases of gastric dilatation in tetany.

If one were to judge by such experiments as those of Falta, cited above, or be guided by therapeutic results in the case which I report, perhaps the conclusion might be reached that gastric tetany is only an incident, sometimes present and sometimes absent in the general condition of chronic tetany.

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It may at least be suggested that no patient should be operated on for dilatation until proper remedies have been used for balancing vagotonic and sympatheticotonic conditions.

J. S., woman, aged 26, single, first examined Aug. 8, 1914, had negative family history except that one sister was a great sufferer from bronchial asthma. Personal history was negative. The present illness began in the latter part of 1913. After suffering for some time with dyspepsia, the patient had an acute attack of indigestion which was followed by some sort of convulsion. After this, it was noticed that she had apparent hemiplegia of the left side. At infrequent intervals these convulsive seizures returned, and general prostration became very marked.

At present examination, patient is not able to walk without assistance, and the indigestion has been so pronounced that she has been using stomach pump and lavage every day.

It was some time after this examination that she was seen in one of these so-called convulsions. This came one day after a bath. This convulsion lasted several minutes, in fact was not relieved until sufficient chloroform was used to produce thorough relaxation. She appeared to be thoroughly unconscious, but it was afterward learned that she could hear and understand some things that were going on. The arms were sharply flexed, the hands closed and pressed so strongly against the neck as to interfere with respiration. Respiration was somewhat labored, but not changed in rhythm. The legs were rigid and cataleptic.

The nature of this attack, together with other symptoms and signs, left no doubt as to the diagnosis of chronic tetany.

Briefly, these signs and symptoms were as follows:

1. Bilateral spasms of the hands, lower extremities and respiratory muscles, as already described.
2. General muscular weakness and partial hemiplegia of the left side.
3. Gastric dilatation.
4. Transverse ridges on the teeth.
5. Chvostek's sign.
6. Trousseau's sign.

These in themselves appeared sufficient basis for the diagnosis of chronic tetany. At this stage of my study and treatment of the case, Dr. Barker of Hopkins happened to be in our city and gave a very interesting clinical lecture on this case, confirming the diagnosis.

It became an interesting study, however, in the further management and treatment of the case to note the symptoms referable to the autonomic nervous system.

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In Van Oorden's tabulation of sympatheticotonic and vagonic symptoms, he presents in parallel columns about thirty-six symptoms in each group opposing or balancing each other. In this case, there were only two that belonged to the sympatheticotonic, and these were not present very long, namely, urticaria and low gastric acidity.

On the other hand, there were present, more or less all the time, except when relieved by treatment, about twenty vagotonic symptoms, namely: myosis; frequent winking; frequent spitting; arrest of secretion of gastric glands; hypermotility of stomach and intestines; vomiting; enophthalmos; bradycardia; low blood pressure, 90 systolic; asthmatic attacks occasionally; pylorospasm; dermatographia; gag reflex absent; clammy hands and feet in later stage of case; eosinophilia in early stages; increased fat tolerance; increased sugar tolerance both before and after epinephrin; pilocarpin caused extreme salivation; atropin well borne; epinephrin well borne.

The patient's response to the therapeutic tests of epinephrin, atropin and pilocarpin was very instructive. A very small dose (1/20 grain) of pilocarpin produced excessive sweating and salivation. Epinephrin chlorid, hypodermically, was well tolerated, and on account of its quick action, more than once prevented a threatened convulsion. Atropin also was well borne, and by its use, pylorospasm was relieved, after which the gastric dilatation disappeared, as shown by roentgenogram, all of which would indicate that in this case we have not only deficient parathyroid secretion, but also a disturbed condition of the autonomic nervous systems.

Treatment.—Diet: The diet indicated in such cases consists of foods rich in calcium, as milk, eggs, etc. In this case no material improvement was obtained until diet had been limited to buttermilk. Later eggs, fish, chicken and vegetables were added.

Parathyroid gland tablets, from 1/20 to 1/10 grain each, have been given continuously and seem to be advantageous, but they are not of themselves sufficient to control the symptoms.

The powdered suprarenal gland is given in capsules, each of 5 grains, three times a day, and seem indispensable. In threatened convulsions, epinephrin chlorid, from 0.001 to 0.002 gm., usually wards off an attack. Under use of suprarenal powder, blood pressure was brought up from 90 to 110.

Powder of the pituitary body adds to the comfort of the patient and is given almost continuously in 5-grain doses. It does not seem so indispensable as the suprarenal powder.

Calcium lactate has been given in 20-grain doses three times a day and is indispensaable. Once, when calcium lactate could not be obtained at the drug store, calcium chlorid was substituted temporarily. It may have been a simple coinci-

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dence, but a convulsion followed a half hour after the first and only dose given, indicating, as some assert, that chlorids are harmful.

Atropin, 1/100 grain, given hypodermically, gave first relief to pylorospasms, and under its use for a short time the gastric dilatation gradually disappeared.

Later, tincture of hyoscyamus, in full doses, was given for a period of two weeks.

The patient had been using lavage daily. I gradually dispensed with this after beginning atropin.

Massage and electricity were used regularly while the patient was in the hospital. She was dismissed April 4, 1915, very much improved.

One complication surprised me, and I have not seen it mentioned in connection with any other case, namely, hemorrhage, apparently vicarious menstruation. The first hemorrhage came from the pharynx. A capillary oozing for several hours resisted all local and general treatment, until atropin was given, which arrested it promptly. This hemorrhage was estimated to have amounted to 2 or 3 pints, and caused extreme anemia and prostration lasting several weeks. There has been a second hemorrhage, occurring also during menstruation, since the patient left the hospital, this time from the bowels. I had neglected to give instructions to use atropin, and again a large amount of blood was lost.

The patient is still under the treatment outlined, and I do not consider her cured. A recent letter, however, says that she is doing well and hopes soon to go to the mountains.

ABSTRACT OF DISCUSSION

Dr. J. T. Fisher, Los Angeles: In regard to this interesting report of a chronic tetany, Dr. Munroe has made two points which would lead me to think that perhaps in addition to tetany the patient was suffering from some functional disturbance which might be designated by some of us under a different heading. I wish to ask Dr. Munroe if, in his examination, in addition to the points he brought out, he determined whether or not the patient had limitation of the visual field or any sensory defects that we find in ordinary hysteria? The case at least had some of the ear marks of that most protean psychosis.

Dr. Julian M. Wolfsohn, San Francisco: I am glad to see that Dr. Munroe has attacked this important subject of tetany from the standpoint of the vegetative nervous system. So far there are very few cases of tetany that have been worked up with special reference to the vegetative nervous system. Using as a basis different tests, in one case of tetany that came under my observation, which I have described in The Journal of May 16, 1914, I have come to

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practically the same conclusion as Dr. Munroe. The majority of symptoms fall on the vagotonic side of the classification. An important keynote to the treatment of the case is thereby gained. It would be very interesting to have more reports, especially from the surgeons, who occasionally see artificially produced tetany, and from the clinicians who see these patients more often.

Dr. Cecil E. Reynolds, Los Angeles: Can Dr. Munroe give us the physical signs of the hemiplegia that he spoke of? I know nothing about hemiplegic tetany, but its presence in this case certainly suggests a hysterical element. In many cases, when there is pylorospasm, this complication is so extremely distressing and lowers the patient's resistance to such an extent as to make manifest in a physical form deep emotional complexes preexisting in the mind.

Dr. Josephine A. Jackson, Pasadena, Calif.: I wish Dr. Munroe would tell us the whole story of this subject. Dr. Reynolds, Dr. Fisher and I on this western coast are inclined to view this case as hysteria. I do not recall any symptoms, as Dr. Munroe read his paper, that would be lacking in hysteria. The physical symptoms and loss of faucial reflexes might be present in hysteria. The contraction of the muscles, the obstetric hand, the overexcitability of the various reflexes, I find again and again in cases of hysteria. The cardiospasm and pylorospasm are exceedingly common in hysteria. The fact that the patient had this condition, marked at times, and then disappearing (elicited by pressure on the forearm) would suggest that the very attention that was paid to her in eliciting the symptom intensified the tonicity of her muscles. An increased excitability of the reflexes may obtain in conditions as widely different as that of hysteria from the normal condition of a parturient woman. All obstetricians know that if the woman is afraid of her physician, if she has no confidence in him and feels that he is some terrible bringer of instruments, all uterine contractions may cease when he comes into the room. The cause is purely psychic, of course, but it is sufficient to check the hormone formation that stimulates the lumbar cord which brings about contraction of the uterine muscles. The hysterical individual likes an audience, and must have attention. If this woman had the obstetric hand when she did not know she was observed it would more readily suggest tetany than having it only when being tested. The doctor described wonderfully well the influence of the vagotonic and sympathetic nervous systems. We are just touching the edge of knowledge of this subject. The interrelation between the influence of the psyche (mind) on this machinery of its own, and the influence of this machinery on the mind that is the master, is so close that we can hardly tell which inaugurates the activity of the other.

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DR. FRANK R. STARKEY, Philadelphia: That tetany is at least at times hysterical in nature I am convinced, although the rôle of the autonomic nervous system and the relation between the autonomic nervous system and the glands of internal secretion would lead one to believe that these are involved in many cases. One of the cases which rather vividly impressed on me the degree of suggestibility that sometimes exists in this condition was that of a man who had been treated at a hospital in Philadelphia for a number of years for attacks of tetany. If he were pressed on the biceps or the foot on this side was markedly extended, he would immediately go into a very painful tetanic spasm. He was so minute in the description of his condition that I was satisfied suggestion had a good deal to do with it. I talked with him along the lines of complete recovery and said to him: "I have something here that will surely either completely cure this condition or greatly benefit you at once." I gave him 1/10 grain of calomel by mouth three times a day and he was completely well within forty-eight hours. That was four years ago and there has never been a return. It must have surely been a purely mental condition in his case and suggestion brought about recovery.

DR. GEORGE A. MOLEEN, Denver: I think to separate the dilator fibers of the pupil from the sympathetic system may possibly be an error. I think it has been rather clearly shown that the dilator fibers take their origin in the first and second sympathetic, which supply the auxiliary spinal system (ciliospinal) to be delivered to the radiating fibers through the ophthalmic branch of the gasserian ganglion. This is supported also by two or three pathologic conditions. Tumors of the gasserian are frequently followed by paralysis of the dilator fibers, as well as the same result in enucleations of the ganglion. Also the tuberculous glands of the neck which we see so frequently in Colorado have given rise to many theories for the anisocoria or unequal pupils seen with the dilatation on the same side. The character of the muscle fiber as well is sufficient to indicate its sympathetic supply. It is pretty well established that the dilator fibers like the circular fibers receive their supply from the sympathetic system. This is merely a matter of comment on what I understood Dr. Munroe to have said as to the supply of the pupillary fibers.

DR. J. P. MUNROE, Charlotte, N. C.: Dr. Reynolds asked especially about the hemiplegia. As a matter of fact, it was not a true hemiplegia. On examination I found that the weakness was general. There was no Babinski and it was not a true hemiplegia at all.

As to the suggestion that it may have been a case of hysteria, I will admit that on first examination I considered it hysteria, and it took me some time to reach a different con-

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clusion. There were no stigmata of hysteria that I could detect. No contraction of the visual field. There was no emotional disturbance. The patient was one of the most even tempered people, always cheerful, and had a very bright disposition. Dr. Barker of Hopkins happened to be in our city when I had made my diagnosis, and he thought at first he had me up a tree, and gave a very interesting clinical lecture on this case before the county society. It was only in the latter part of the examination that he came to the conclusion that my diagnosis of chronic tetany was correct.

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FOCAL DISEASE OF THE BRAIN

A CLINICAL REPORT OF EIGHT CASES

CHARLES R. BALL, M.D.
ST. PAUL

The medical profession in general is inclined to be pessimistic with reference to operations for the removal of brain tumors. If the operation for the removal of a suspected brain tumor is proposed, the surgeon rather delights in quoting to you, with a shrug of his shoulders, that statistics show that recovery takes place in only from 3 to 5 per cent. of all cases. In answer to this general view, Cushing says:

> It depends on what is meant by recovery. If we put a strict construction on the word recovery, the statistics of the surgeon are probably correct, but if we can be satisfied with the alleviation of suffering, the preservation of vision and the prolongation of life in relative comfort and usefulness, often for many years, certainly 50 or 60 per cent. of all patients can be thus helped.

We are so intent on the localization and removal of the growth in its entirety in order to obtain a recovery in its strictest sense that we often miss the opportunity offered for relief in the 50 or 60 per cent. of the cases. Some of us no doubt are able to recall several humiliating occasions when we thought we had surely localized a brain tumor, but, after the surgeon had exposed the area where we fully expected to see it tower above the surrounding brain tissue, nothing but normal brain in all directions was found. The tumor, the chief object of the operation, from our point of view, was not found, and in our disappointment we forgot all about the relief of symptoms which, from the patient's point of view, was the

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real reason why he submitted to the operation. If we cannot find the tumor, let us try at any rate to do everything possible to afford our patient relief.

Case 1 is a good illustration of what may be done toward this end although no tumor was found.

CASE 1.—Mr. G. S., aged 50, was seen in consultation in October, 1913. His symptoms were those of a slowly developing right-sided hemiplegia, the paralysis beginning in his foot and within a period of six months involving the entire right side. In addition to the paralysis, he had very severe attacks of epilepsy about once a week. These attacks were increasing in frequency. He had great fear of these attacks, and for several days after an attack he was quite confused mentally. There was nothing of a jacksonian character about the attacks. He was greatly depressed and cried easily. Six years before the beginning of his trouble he had fallen downstairs and injured his head quite badly.

Examination.—The serum Wassermann and spinal fluid examinations were negative. He had previously taken specific treatment to which he did not respond. The deep reflexes on the right side were more active than those on the left. There was also a Babinski. There were no sensory changes. The eye backgrounds were normal.

The diagnosis of brain tumor, involving the left motor area, was made and an operation advised with the understanding that it was not probable the tumor would be found, but that the outlook for relieving the epilepsy was good. The patient consented to the operation because of his great desire to be relieved from his attacks of epilepsy.

Operation.—The left motor cortex was located according to the method of Krönlein; a large bone flap was made over this area and the brain exposed. The appearance of dura and cortex was fairly normal. By palpation of the brain with the finger no evidence of any tumor lying beneath the cortex was discovered. With the firm conviction, however, that somewhere in the left motor area there must be a lesion which by reflex irritation through the left motor cortex caused the epileptic seizures, it was decided to clip out the cortex according to the method of Feodor Krause. The cortex was accurately located by means of faradic stimulation, and the entire area thus determined was removed to the depth of about one-fourth inch.

Postoperative History.—The entire right side was completely paralyzed for about two weeks after the operation. Then gradually the strength in it began to return. The epileptic attacks disappeared entirely. A recent inquiry in regard to this patient brought the information that he was

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in excellent health and spirits and was able to take care of his business. Some paralysis still existed on the right side, but he had better use of his leg and quite as good use of his arm and hand as he had prior to the operation.

The removal of the motor cortex in this case saved the operation from being a complete failure. With no visible tumor to remove, we endeavored to do the next best thing and relieve our patient of his most distressing symptoms.

CASE 2.—Mr. G. S., aged 40, was seen in consultation Oct. 7, 1914; family history negative; personal history unimportant. Patient was perfectly well until in January, 1914, when he began to suffer with headache. The headache was more or less constant and had gradually become worse; when I first saw him it was so severe at times that full doses of morphin hypodermically failed to relieve him. During these severe headaches he became delirious. He occasionally had attacks of vomiting, projectile in character.

Examination.—The serum Wassermann and spinal fluid examinations were negative. The intraspinal pressure was greatly increased; temperature was normal; white blood count, 14,300; slight trace of albumin in urine; physical condition fairly good; hemoglobin, 79 per cent. There was considerable pain in back of head extending down the neck; any jarring, as riding in train or shaking the bed, increased discomfort in head. A good deal of rigidity in the muscles of the neck was present. Percussion in the occipital region seemed to cause more pain than elsewhere on the cranium; changing position from lying down to sitting up or moving the head, caused dizziness. There was no nystagmus or paralysis of eye muscles, There were double choked disks on both sides with retinal hemorrhages. Hearing in both ears was good. The two sides of the face seemed equally well enervated. Deep and superficial reflexes were present on both sides. There was no Babinski. There were no sensory disturbances. When lying down patient appeared to have good control of both upper and lower extremities. When he walked, he threw feet forward and body and head backward, so that he had to be well supported to keep him from falling backward. Vigorous specific treatment gave him no relief.

As there were no definite localizing symptoms and the headache was becoming unbearable, a decompression operation was advised. Taking into consideration the occipital pain, rigidity of neck muscles, high degree of choked disks, vertigo on change of position and the position assumed by the patient when he attempted to walk, the good mental con-

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dition of the patient, except when he was suffering severely, and the absence of any paralysis, it was thought wiser to make the decompression in the occipital region.

Operation.—Suboccipital decompression was performed on October 17, exposing fully both lobes of the cerebellum and the medulla; the dura was very tense and when it was opened serous fluid oozed out. The cerebellum presented a normal appearance; the cerebellar-pontine angle on both sides was exposed and a trocar was put into the cerebellar lobes in different directions, but nothing was found. The dura was left open and the wound was closed.

Postoperative History.—The patient made a fairly uneventful recovery from the operation. His relief from severe headache was immediate. In two weeks his choked disks had entirely disappeared. Up to the present time he appears to be in the best of health and says he never felt better in his life.

The case may be regarded as an unusually successful result from decompression. It also illustrates a point which I wish to emphasize. Owing to the complexity of brain function frequently we are unable to localize a brain growth accurately. If, however, we study all the symptoms with reference to whether the growth is situated anterior or posterior to the tentorium, we shall be less apt to err in our decompressive measures than if we attempt a more definite localization. We are sometimes misled in cases in which the general symptoms all point to a posttentorial growth but because of some few paralytic symptoms direct our efforts to the motor area.

CASE 3.—Miss M. S., aged 29, single, schoolteacher, was seen in consultation Dec. 5, 1914. Family and personal history, unimportant. The present trouble began seven years ago with a slight buzzing and gradually increasing deafness in right ear. The past two months she had attacks of vomiting of the projectile type. She has been able to teach regularly, however, until within three or four days of the date of the consultation. She now complains of headache, which has been present for some time. This is particularly severe in the back part of her head and down her neck. The pain is increased on coughing or sneezing as well as by any other jarring, such as riding on the train. There is a feeling of stiffness in the muscles of the neck.

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Examination.—General state of nutrition, good; heart and lungs, apparently normal. Station is poor, with tendency to fall toward the right; in walking her gait is unsteady and she tends to fall, also, toward the right. Pupils symmetrical, equal in size and reacted to both light and accommodation; horizontal nystagmus, areflexia present in right eye, left eye normal. There was diminished sensation on the right side of face and right side of tongue. There was a slight involvement of the seventh nerve on the right side. Hearing in right ear greatly diminished; left ear, normal. Bone conduction in right ear not increased. No other involvement of the cranial nerves was observed. The deep reflexes were normal. There was no Babinski. Loss of muscle sense in right arm and leg was marked. There was an awkwardness and slowness in the movements of the right arm and hand (adiadokocinesis) apparent in supination and pronation and movements of the fingers. Percussion over the head did not reveal any particular area of sensitiveness; mental condition, good; serum Wassermann, negative; urine, negative; choked disks on both sides, with only slight impairment of vision.

The diagnosis was slow growing tumor of the right cerebellar-pontine angle. Operation was advised and accepted.

Operation.—A tumor about the size of an English walnut was found in the right lateral recess and as much of it as possible removed. It was found to be sarcomatous in character.

Postoperative History.—The recovery of the patient after operation was uneventful. A recent communication from her physician says: "Two days ago she came alone to my office and walked up a long flight of stairs. She walks as yet in an unsteady manner and does not dare to trust herself away from home in any walks of more than three or four blocks. There is still some difficulty in the use of the right hand but this is better than before the operation. The headache and choked disks have entirely disappeared and the vision is normal in both eyes. There is just a suggestion of the nystagmus; sensation on the right side of the face is normal but still diminished in the tongue. In general her condition has steadily improved since the operation, although the improvement in the past two or three months has been very slight. She is planning to take up her school work in September."

This patient is another example of a partial recovery, since the nature of the growth and the fact that it was not entirely removed makes the outlook for its return extremely probable. It cannot be classified in the statistics of absolute recovery (from 3 to 5 per cent.), but may justly be regarded as a success-

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ful brain operation, since relief from all distressing symptoms up to the present time was obtained. The case is interesting because of the length of time intervening between the appearance of the first symptoms and the development of symptoms severe enough to cause the patient to seek medical aid; also for the reason that she was able to continue her regular work as schoolteacher for so long a time with such a large growth in this location.

CASE 4.—Mrs. M. W., German, housewife, married, aged 52, was seen in consultation March 4, 1913. Family history and personal history, up to beginning of present trouble, unimportant. Present trouble began about one year previous to date of consultation with a severe attack of epilepsy in which the patient fell to the ground and was unconscious for several hours. She has had two attacks of this nature about a month apart. In addition to these attacks she has seizures coming three or four times weekly which are distinctly jacksonian in character. The attack always starts in the left thumb with a cramping and painful sensation; this rapidly extends to the forearm, then arm and face and sometimes to the other side of the body. During the attack the pain in the muscles of the arm is intense. Usually consciousness is retained but occasionally it is lost for a few seconds. The entire attacks last three or four minutes. Her memory is poor, particularly for recent events; she becomes dizzy when she gets up quickly or bends forward; stooping down will often bring on an attack.

Examination.—The physical condition of the patient is good. She eats and sleeps well. She is quite free from headache, and percussion over the cranium does not reveal any sensitive areas. The pupils are symmetrical in shape, but the left is larger than the right; both react to light and accommodation. The eye backgrounds are normal. Movement of bulbi free. There is no nystagmus. Hearing and vision good. No paralysis of facial nerve. Deep and skin reflexes present. No Romberg. The sensation, for both touch and pain, is much diminished in left hand. Astereognosis in left hand. She is quite clumsy with her left hand. The strength in it is also lessened. Serum Wassermann negative. No spinal fluid examination was made.

The diagnosis was brain tumor situated in right posterior central convolution.

Operation.—This patient was operated on April 1, 1913. A large flat tumor covering and firmly adherent to the brain cortex, lying mostly posterior to the fissure of Rolando was

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found. This tumor had perforated the dura in many places and had eaten into the skull. Because of its great tendency to bleed, its firm adherence to the brain tissue beneath it and its evident malignant nature, no attempt was made to remove it. Microscopic examination of small portions of it removed at the operation indicated that it was of a sarcomatous nature.

Postoperative History.—The patient lived almost two years after her operation without much change in her condition.

In this case it is interesting to note that the symptoms correspond with our ideas of brain localization; also, notwithstanding the presence of a large tumor which had in many places destroyed the dura and bone tissue about it, there was almost complete absence of pain and no sensitive areas were discovered by percussion.

CASE 5.—Mr. N. C., Swede, aged 60, was seen in consultation Sept. 15, 1914. Family and personal history not important. The patient says he has always been in excellent health. He cannot remember ever having had a severe illness. No history of influenza or ear disease. He was feeling perfectly well and working on his farm, as usual, when one day, six weeks before his visit to me, he was suddenly seized with a severe epileptic convulsion and fell unconscious to the ground. He remained in an unconscious condition for several hours. He apparently made a good recovery from this attack, but in three or four days he had another of the same nature, and after this attack he noticed an awkwardness and clumsiness in the use of his left arm and leg. This condition of the left side had steadily become worse so that at the time of examination paralysis of the left leg was almost complete. The patient also had typical attacks of jacksonian epilepsy, starting in the left toe and extending over the entire left side. The slightest irritation of the skin over the left side would bring on one of these attacks. Occasionally he lost consciousness in these attacks but usually not. He complained of considerable headache, mostly over the back part of his head.

Examination.—Physically, nothing significant was observed except a marked degree of pyorrhea. Temperature was constantly normal. Pupils were equal and reacted to both light and accommodation. There was no nystagmus, no cranial nerve paralysis. The eye backgrounds were normal. The deep reflexes in the left side were more active than in the right. Ankle clonus and the Babinski were present in the left side. The muscle sense on the left side was greatly

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disturbed. The patient when asked to close his eyes and touch his nose with the tip of his index finger seemed unable to locate his face. He was unable to recognize any object put in his left hand by the sense of touch. His mind remained clear until within a few days before his operation, when he became mildly delirious at times. The entire cranium was sensitive to percussion but apparently no one place more than another. Urine negative and serum Wassermann negative.

Operation.—The patient was operated on Oct. 3, 1914. The brain over the right motor area was exposed. In breaking back the bone flap the dura was torn and on inspection a little pus was noticed oozing out through the opening; tracing the pus back to its origin, a large completely encysted abscess was found situated about one-fourth inch below the dura, situated at the upper part of the Rolando fissure and posterior to it. The abscess contained about 2 ounces of white thick pus.

Postoperative History.—The patient recovered from the immediate shock of the operation but died on October 7, four days afterward, of septic meningitis.

The interesting feature in this case was the unexplainable cause of the abscess, unless the pyorrhea might be so regarded and the evidently long duration of it without causing any symptoms.

CASE 6.—Male, German, aged 48, was seen in consultation Oct. 5, 1914. Family and personal history unimportant except that patient was kicked on the head over the left parietal region when 11 years old, by a horse. He was not rendered unconscious by the injury nor was he attended by a physician. He remembers that his mother dressed the wound and it discharged pus for some time. The scar is still plainly visible. In August, 1914, he tripped and fell on a hard cement floor, striking his shoulder and face and jarring his whole body. A few days after this fall he felt some pain in his big toe on the right side. He soon began to notice that he could not handle the right leg and foot as well as formerly. This condition increased and gradually involved his arm. At the time I first saw him he could not walk without help and then with the characteristic swing and drag of the right limb, typical of a hemiplegic condition. He complained of headache chiefly in the occipital region.

Examination.—His mentality was good. He had no vomiting spells. His vision was also good. Nothing significant was found in the physical examination; temperature normal; serum Wassermann, negative; spinal pressure, much increased. Globulin reaction ++. Lymphocytes 5/3.

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Spinal fluid Wassermann, negative. Pupils were equal and reacted to both light and accommodation. No nystagmus. Field of vision, normal. Eye backgrounds, normal. No evidence of cranial nerve involvement. No Kernig. Both deep and superficial reflexes present. No ankle clonus or Babinski; slight Romberg; no sensory disturbance; marked weakness of right arm and leg.

Patient was able to be up and walk around with help and on several occasions went out for an automobile ride; each time after returning he seemed more helpless. On October 10 he began to vomit and became stupid and confused mentally. The paralysis in arm and leg was now absolute.

Operation.—Oct. 13, 1914, he was operated on. A large bone flap was made over the motor cortex on the left side. When the dura was opened no pulsation could be felt, and an area of boggy consistency was revealed. When a trocar was put into this area a clear yellowish fluid flowed out and on enlarging the opening numerous fresh blood clots were found. This cyst was situated between the dura and brain and was flattening the brain convolutions down into the cranial cavity. The walls of the cyst were as thick as the dura itself. The cavity of the cyst was packed with gauze and the wound closed.

Postoperative History.—The next day after the operation the patient began to use his paralyzed arm and leg apparently without difficulty. He was delirious for several days and had several convulsive attacks; otherwise his recovery was uneventful. At the present time he appears perfectly well and works every day.

This case is interesting in that it shows the remote effects of old head injuries. The cyst was undoubtedly caused by the injury which the patient had received when a boy. It was probably beginning to degenerate when he had his fall in August. This fall started the cyst to bleeding and enlarging. The automobile rides increased the bleeding and consequently the rapid progression of symptoms during the last week before the operation were explained.

The two following cases are examples of disease of the pituitary body:

The following case falls in Dr. Cushing's classification of pituitary disease in Group 1, representing those cases in which, along with definite constitutional disturbance, caused by defective glandular function, there are also neighborhood symptoms due to pressure.

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CASE 7.—Male, aged 19, family history, negative. This boy was a full-term baby. He was normal in every way up to his eleventh year; at that age he began to lose his hearing, first in the left and then in the right ear, and gradually within a period of three years he became totally deaf. No other symptoms accompanying this deafness were observed. In July, 1914, five years after he had become totally deaf, he was taken suddenly sick with vomiting and severe pains in the left temple. The severe pain continued for four or five days, when a complete ptosis of the left eye occurred.

Examination.—When I saw the patient in August there was still the ptosis, a dilatation with paralysis to both accommodation and light of the left pupil, and some neuralgic pain at times. No other symptoms were observed. The Wassermann in the blood and the three reactions in the spinal fluid were negative. No satisfactory diagnosis was made at this time. On Feb. 8, 1914, I saw this patient again. The ptosis had almost completely disappeared, the pupil reacted to both accommodation and light and was nearly normal in size. In the seven months since my last examination the patient had gained 15 pounds in weight; his lips were somewhat thickened and protruding. His voice was falsetto in character. There was a development of his breasts and a rounding out of his hips which suggested the feminine type of form. About one month before his visit to me he began to lose the vision in his left eye. An examination of the field of vision showed vision in the left eye almost entirely gone. He could barely distinguish as shadows one's fingers on the nasal side; on the temporal side he was totally blind. In the right eye his nasal vision was good, but on the temporal side it was gone. There was also a paralysis of the external rectus. The eye backgrounds were fairly normal in appearance. Very little pain was complained of. There was 0.5 per cent. of sugar in the urine and a mild polyuria. He got up about three times during the night to pass his water. A roentgenogram (Fig. 1) of his cranium showed an enormously enlarged sella, about 3 cm. in width and 2 cm. in depth, with the posterior clinoid process scarcely visible. The anterior wall of the sella appeared to project into the sphenoidal sinus. A diagnosis of tumor of the hypophysis was made and operation advised.

Operation.—This was performed on February 27, by Dr. Cushing. A hemorrhagic cyst situated in the center of the pituitary body was found; the contents were evacuated and the walls destroyed as well as possible.

Postoperative History.—The patient made an uneventful recovery. On April 24, less than two months after the operation, vision in the right eye was fairly normal, and in the left eye, fingers on both the nasal and temporal sides were easily counted by the patient. His voice had lost its falsetto

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character. Sugar was still present in the urine but in less than half the amount found before the operation. The neuralgic pain and polyuria had disappeared entirely.

CASE 8.—Male, aged 14, family history, negative; personal history, up to time of present trouble, unimportant. In January, 1913, the patient received a severe blow on the right side of the forehead, just above the right eye, which rendered him unconscious for about ten hours. As a result of this blow there was a cut above the right eye about 1 inch in length, the scar from which is still plainly visible. On the day following the accident he complained of an attack of dizziness which lasted fifteen or twenty minutes, during which the objects in the room seemed to go around and he lost consciousness for a few minutes. The attack was followed by a headache. At first he had similar attacks every day, but after a few months they became less frequent, occurring at the present time two or three times a week. In these attacks he must always lie down or sit down. In the dizzy part of the attack he complains of a noise in his ears, which he describes as like the sound of a bell after it has quit ringing. In addition to the dizzy attacks he has periodic attacks, coming regularly once in three or four weeks, of severe headaches during which he is completely prostrated and vomits frequently a greenish fluid. The resemblance of these headaches to migraine in a patient with such typical symptoms of hypopituitarism leads one to wonder whether disturbed function of this gland may not play an important rôle in the causation of migraine in general. The patient's disposition is irritable. He has had two severe nosebleeds since his accident. He goes to school almost every day but usually comes home at 10 o'clock recess on account of headache. He has made no progress in school whatever since his accident although previous to the accident he made good progress. He has not cared to play or indulge in games with other boys. His appetite in general is poor. The patient's father and mother, as well as his brother and sister, are slender. Several days before his accident, dressed in a heavy sheep-lined coat he weighed 92 pounds; in May, 1913, his first weight taken after the accident, he weighed 114 pounds; on Dec. 29, 1914, 169 pounds; in March, 1915, 181 pounds, and in April, 194 pounds, making a gain of over 100 pounds in a little over two years.

Examination.—The physical and neurologic examination is in general negative. Equal parts of urine with Haines solution has at times given a complete reduction, with a reddish yellow deposit. The roentgenograms show a slightly enlarged sella, the width of which is 15 mm. and the depth 12 mm. The general appearance of the patient at first glance bears some resemblance to the myxedematous type. The face is anemic and puffy; the lips thick; the breasts and hips are

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Fig. 1.—Greatly enlarged sella in Case 7.

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Fig. 2.—Slightly enlarged sella in Case 8; posterior clinoid indistinct.

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Fig. 3.—Patient 8, showing tendency toward obesity and feminization of type.

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greatly enlarged; the abdomen is protruding and in standing erect the limbs assume the knock-knee position observed in the female. The skin presents a mottled bluish appearance in many places. The hand is typical of this type of pituitary disease, the fingers tapering to very small end phalanges.

This case belongs to the third group in Cushing's classification, in which the neighborhood symptoms are wanting and the constitutional symptoms are predominant. These cases are especially interesting because they call attention in a striking manner to the remarkable constitutional changes which may be produced in a comparatively short time by the disturbance of functions of this little gland. When we reflect that this gland is only one of a number which seems to be intimately related in function and vitally important to our physical and nervous welfare, we are better able to realize the complex nature of life and the enormous difficulties which often confront us in accounting for symptoms and making diagnoses in so-called functional nervous disease. The number of cases reported is too small to permit of any general deductions. I wish to emphasize, however, two points mentioned in my introduction. First, the result in operations for brain tumors would be more often successful if our localization was studied more with reference to whether the growth was situated anterior or posterior to the tentorium. If this can definitely be determined decompression can be made to relieve the pressure symptoms at least for a while. Second, if no tumor or signs of tumor are found, let us not throw up our hands immediately, but endeavor in a rational and intelligent manner to ascertain if something further may not be done for the relief of the most trying symptoms.

Lowry Building.

ABSTRACT OF DISCUSSION

DR. C. EUGENE RIGGS, St. Paul: Dr. Ball's paper recalls to all of us many interesting cases of focal brain disease. Among the number of hypophysial growths I have seen, I

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succeeded in referring one to Dr. Cushing. He operated; there was marked amelioration of symptoms for a year, with subsequent death. I have come to the conclusion that when epilepsy manifests itself after middle life, it is suggestive of syphilis or brain growth. I had a case, apparently of essential epilepsy—a young man—on going into the history of which I found that a year previous there had been trauma of the cranium. An exploratory operation was made and we found a cyst overlying the cortical area. Last year the *British Medical Journal* contained a reference to some thirty cases of brain tumor in which the symptoms had been absolutely masked by a psychosis. Recently I examined a case of Dr. Hammes' in which the patient apparently was suffering from a manic-depressive state. It was only toward the close of his life that any objective symptoms manifested themselves, and postmortem revealed a large growth.

Dr. Cecil E. Reynolds, Los Angeles: I was surprised to hear that in Dr. Ball's second case the surgeon undertook a subtentorial decompression without any localizing signs. I gather from Dr. Ball's paper that there was nothing to show any lesion under the tentorium, and I cannot see why that most excellent operation of Cushing—the subtemporal decompression—was not done in that case. The subtentorial operation is inconvenient from the standpoint of gravity, and, as Horsley pointed out many years ago, the danger of all brain operations increases as you approach the occiput and as you get near to Reid's base line. Personally, I think by far the greatest danger of operations for tumor is in the pressure. I do not think one can overestimate it. In all cases in which there is intense headache, vomiting and raised blood pressure, etc., I would do a subtemporal decompression before attempting to look for a tumor. The case of brain abscess without assignable cause was particularly interesting to me. I operated in a similar case and reported it in The Journal in 1912, and Henderson reported a similar case about the same time in the *Lancet*. The case of jacksonian epilepsy which was cured by excision of the center was a particularly fortunate one. I have never been bold enough to cut out a center that appeared healthy to the naked eye, although in one case recently of jacksonian epilepsy in which the fits began in the hand, I found no localized lesion, but by my somewhat thorough palpation of the hand center I caused a paralysis of the arm below the shoulder which lasted for some weeks. During this time there were no fits, and I began to think that the child would remain well, but, unfortunately, when the paralysis cleared up the fits recurred. It is possible that if I had been as radical as was Dr. Ball, a permanent cure might have ensued.

Dr. D. M. Hansen, Lincoln, Neb.: I would like to ask whether Dr. Ball used the Roentgen treatment for hyper-

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pituitarism. I think the majority of patients would consent to that more readily than to operation.

DR. G. A. MOLEEN, Denver: I do not think there is anything more flattering than finding a tumor where one has, through the study of localizing symptoms, presumed it to exist, and nothing is more elusive than a diagnosis based on what seemed to be certain symptoms. Sometimes radiographs may assist us, but only when we take very complete pictures. They should be taken from both sides and anteroposteriorly, and preferably stereoscopically. A good deal of accent is given to a picture if some of the bony configurations which should show through are blurred at a certain point. The more dense structures usually give a distinct shadow and therefore dense tumors should usually give a very striking shadow. On the other hand, I have had in my experience a cyst giving a distinct shadow so large that it was thought to be a defect in the plate, and yet when the cyst was opened on the operating table it was found to occupy almost the entire hemisphere. In a recent case of tumor the skiagram showed a distinct shadow in the neighborhood of the sella turcica. The symptoms were referable to the anterior portion of the temporosphenoidal lobe. There was not sufficient remaining of the optic phenomena to say that there was a bitemporal hemianopsia, since the blindness had already progressed to a great extent, but operation revealed an accumulation of fluid deeply situated in the anterior portion of the temporosphenoidal lobe near the middle line.

In reference to pituitary tumors, I have in mind a case (I do not know whether to say a boy or a man) 4 feet 6 inches in height, 28 years old. Growth stopped at 12 years following an attack of scarlet fever. Skiagram of the head showed a very large sella turcica. There was peripheral, bitemporal limitation of the visual field. His hands were small, there was no pubic hair and he presented an infantile appearance. The epiphyses of the phalanges and metacarpals were shown by the radiogram to be separated or ununited. This was undoubtedly a tumor of the pituitary body, but not with the usual hypertrophy—rather with a sudden cessation of development. I desire principally to emphasize that sometimes in the hands of good radiographers a skiagram (when taken bilaterally and stereoscopically) may be of considerable contributory value, but should not be relied on exclusively.

DR. C. R. BALL, St. Paul: To answer Dr. Reynold's question in regard to the second case as to why I decided to make a suboccipital rather than a subtemporal decompression, this was one of the points I wished to emphasize in my paper. There was pain and rigidity in the back of the neck, extending down between the shoulders, vertigo on changing position, intense headache, a high degree of choked disk; there

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was also asynergia as manifested by the walk of the patient; he threw his body and shoulders far backward and had to be supported in order to maintain his balance. These were the deciding factors which caused me to recommend the subtentorial decompression. I have never had any experience in treating hyperpituitarism with the Roentgen ray. I do not see from the location of the hypophysis how we could hope to affect it without also affecting the surrounding brain tissue. In shrinking up cysts of the pituitary body we would also be liable to shrink up brain tissue, which in most instances I would not consider a very desirable thing to do.

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SPINAL CORD COMPRESSION FROM LEPTOMENINGEAL CYSTS

WITH A REPORT OF TWO CASES

A. L. SKOOG, M.D.
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The two cases which are incorporated in this report, and about which revolves the discussion of the subject of nonparasitic cysts of the leptomeninges of the spinal cord, are found to be quite uncommon, even though a most thorough search of the literature is made. The bulk of the reported arachnoidean cysts have for their etiology either the cysticercus or echinococcus, which means that they are multiple and complicated. Moreover, it is highly important that the simple, nonparasitic cysts like those which I am placing on record be recognized early, so that immediate proper operative intervention may be instituted. Then a fair prognosis may be offered.

A plea for an early recognition of the nature of the trouble is a chief motive for placing before the profession a report of my two interesting and instructive cases.

The first patient was in an extreme paraplegic state when first observed. Operative treatment improved her condition but not in a satisfactory manner. The second patient came under my observation in an earlier stage, and with a prompt operation and good after-care made a much better recovery.

REPORT OF CASES

CASE 1.—The patient (V. J., single, aged 37, tailoress) was referred to me for diagnosis by Dr. Gray, who performed the operation at St. Margaret's Hospital, which she entered

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July 10, 1912. She complained of inability to walk on account of a paralysis in the lower extremities, pain about region of lower ribs, feeling of numbness in the legs, and loss of control for urination and defecation.

Family.—Father is living, aged 87. He is suffering from a recent progressing senile enfeeblement. Mother died of pneumonia at the age of 73. Patient has three living brothers, all well; two brothers died, one at 22 and the other at 44 of tuberculosis. There are two healthy living sisters. One sister died of "heart disease" at 49, and another of tuberculosis at 23.

Past Illness.—Patient has had measles and epidemic parotitis. The menses were established at the age of 15. There has been some dysmenorrhea but no leukorrhea. Since the age of 16 there have been attacks of "stiff neck" lasting two or three days. Seven years ago there was an eruption below the knees without itching. There was some backache and headache at the time, but no sore throat, alopecia nor subsequent pains. Recovery was prompt.

Present Trouble.—The onset dates from about July, 1909, when patient awoke one morning with a feeling of numbness in the right leg. There was much difficulty in walking and no work was done for one week. Then followed a period in which she was able to work but there was present always a numbness and trouble in walking. Sharp pains around the waist and in the lower chest appeared early. Three weeks before entering the hospital the condition grew rapidly worse and the patient became bedridden. The left leg had become severely involved also. A loss of rectal control and urinary retention with a subsequent overflow and dribbling had appeared. Uncontrollable jerkings of the legs had been present a few weeks. There had been no headaches or vomiting.

Examination.—The mental condition was normal and the physical condition good. The head, chest and upper extremities gave normal findings. The patient was unable to move her lower extremities voluntarily. The paraplegia was of the flaccid type. The abdominal muscles were weak. There were no trophic sores. Some muscular atrophy was present. All deep reflexes in the lower extremities were absent. No Babinski or allied phenomena were found. The abdominal reflexes were not present. All forms of sensation on the right side from the tenth dorsal segmental area downward were reduced, epicritic and protopathic greatly, and deep sensation to a lesser extent. The ninth dorsal segment showed some mild sensory changes, and the eighth dorsal very slight. On the left side there was a mild diminution for epicritic and protopathic sensibility from the first lumbar segment downward, deep pain sensibility being well pre-

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served. The diagnosis of a cord compression with a corresponding progressing transverse myelitis and tract degenerations was made. A neoplasm or a cyst was indicated. It was located as extramedullary and probably within the dura. The sensory chart localized the lesion at the eighth dorsal segment with possibly some compression on the seventh. An operation was advised at the fifth, sixth and seventh dorsal vertebrae.

Operation.—A laminectomy at the region indicated was performed on July 12, 1912, by Dr. Gray with Dr. Nesselrode assisting. The dura in this region seemed quite tense and had a bluish color. Opening it revealed an arachnoidean pocket of fluid. Its diaphanous wall was torn through, and following the evacuation there was hardly any of the pathologic structure visible. The contained fluid was clear, having to the naked eye the same appearance as normal cerebrospinal fluid. There followed an escape of much cerebrospinal fluid. The cord had the evidence of much local compression. It was visibly diminished in size at the eighth and ninth dorsal segments. The dura and external wound were closed. There was an uneventful recovery from the operative procedure. During the first few weeks the condition remained much the same except for a diminution of the painful symptoms. The patient left the hospital after three weeks and was lost sight of. From Dr. Nesselrode and by the report of a lay friend in May, 1915, I have gathered that there has been a slow improvement of all the damaged functions. There are no pains. The patient complains of a weak back. She now has control of the urine and stools. The patient can move the lower extremities, but in a feeble manner. She can walk with crutches, or a cane and some additional support. Her general health is good.

Case 2.—This patient (J. H.) was referred conjointly to the surgeon, Dr. Outland, and myself by Dr. O. P. Mills of Grant City, Mo. The diagnostic observations and treatment were conducted at the Swedish Hospital, Kansas City, Mo., where the patient entered on April 22, 1913. She was 42 years old, the mother of three healthy living children, the youngest being 6 years old. One child died of diphtheria at the age of 3. Her husband is healthy. They live on a prosperous farm.

Past History.—There has never been any serious illness or trauma. Some hemorrhoids have been present since the birth of the first child nineteen years ago. Patient has been subject to occasional "sick headaches." History regarding the menses and pelvic organs was negative. The menstrual flow has failed twice.

Present Illness.—The onset dates from about March 1, 1913, when a numb sensation was noticed in the left ankle. The

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numbness increased and had extended upward to the hip in three weeks. It was difficult for the patient to describe this state, which was somewhat vague, with creeping cold and hot flashes in the leg. There was no pain of consequence except when walking. The pain had diminished at the time of this examination. The right leg was involved two weeks later. There was a pressure sensation about the legs and waist much like a tight band. A weakness in the lower extremities soon appeared, which was evidenced by some difficulty in walking. There was some trouble or delay in micturition. There were frequently sudden desires to defecate, and much haste was necessary to reach the stool. About one year ago there were a few pains in the shoulders, and six months ago a slight attack of numbness of short duration in the legs. These may or may not have been the very earliest manifestations of the present disease.

First Examination.—The psychic state, general health, chest and upper extremities were perfectly normal. There were no traces of hysterical stigmata. The urine and blood analyses were normal. All tests of the cranial nerves gave normal findings.

Motor: The gait showed much defect but patient was able to get about without support. It had neither the true flaccid nor the spastic characteristic. The lower abdominal muscles showed a little weakness. Muscles governing all movements at hip, knee, ankle and toe joints evidenced considerable weakness, the smaller muscles in the distal parts having a greater amount of loss of power. The flexor groups seemed to be weaker than the extensors. The left side was slightly weaker than the right.

Reflexes: Abdominal reflexes could not be elicited. The gluteals were brisk. The patellars were exaggerated to a moderate degree, left more than right. Decided prepatellars were presented. The Achilles were likewise exaggerated, right equaling left. There were no clonuses. The toe sign was positive to the Babinski and Oppenheim tests, but in a rather moderate degree.

A marked diminution of epicritic, protopathic and deep sensation was present, beginning at an encircling line about 3 cm. below the umbilical level. From this up to the umbilical line there were undoubtedly some mild sensory changes to touch and pin-prick tests, but responses were often doubtful in this narrow zone. Above the umbilicus all forms of sensation were normal. The sensory changes began at the same level on the right and left. The thermal sense showed a definite loss from the umbilicus downward. There was noticed in particular a retardation of the responses, especially for the hot tubes. The diminution of the deep muscle sense was mild in the lower extremities. Sense of position was much changed in the legs, being more noticeable at the

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proximal joints in contrast with the distal. More than 50 per cent. of the tests at the toe and ankle joints yielded incorrect replies. The tuning-fork vibrations (256) were not perceptible at prominences of the pelvic bones or lower extremities. No vibrations were felt at the fifth lumbar spinous process. They were diminished at the fourth and normal at the third lumbar.

Second Examination (May 2, 1913).—The subjective symptoms had remained the same or possibly became more prominent. There had been some mild complaints of stomach and shoulder pains. The objective findings compared with the first examination, were entirely pronounced in every particular.

Rachiocentesis was performed on May 4, 1913, at the third intervertebral lumbar space. The pressure registered 350 mm., water gage, and with the patient in a sitting posture. After 30 c.c. were withdrawn the pressure was reduced to 260 mm. The fluid was clear, containing no flocculi. The total albumin content was slightly increased. Likewise there was shown a very feeble positive globulin content by the Nonne-Apelt and butyric acid tests. The Wassermann was negative. The lymphocyte (small and large) count was 12 per cubic mm. There was some débris, and amorphous and crystalline bodies. A number of organic bodies taking a rather heavy stain and appearing to be degenerated epithelial cells, were found. No parasitic organism of any kind, nor tumor cells could be demonstrated.

The diagnosis eliminated syphilis, in particular that of the meninges, also tuberculosis and other rarer chronic meningeal infections. A motor and sensory paraplegia makes it quite evident that there is organic disease or a compression at the lower dorsal spinal cord. A chronologic survey of the development and progress of the symptoms and examination findings invites the diagnosis of an extramedullary new growth or cyst with its upper pole surely at the eleventh dorsal cord segment and probably very mildly involving the tenth dorsal. The lower pole could not be located definitely. The lesion has an intradural location. I stated that we were dealing with a cyst of the inner meninges or a very soft benign neoplasm. There were no hopes for checking the disease otherwise than by a surgical intervention. Accordingly I demanded an immediate laminectomy at the seventh and eighth dorsal vertebrae.

Operation.—The laminectomy was performed on May 7, 1913, by Dr. Outland, assisted by Dr. Clendening. After removal of the seventh and eighth dorsal laminae, there was seen, at the depth of the entire cavity produced, a decided bulging of the dura which was quite tense to palpation. There was diminished transmitted pulsation of the dura from the cord, and it had a passively congested appearance. The

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sixth dorsal lamina was then removed. The dura was cautiously opened, and a considerable amount of cerebrospinal fluid free in the subarachnoidean space escaped. Then there was seen distinctly a cyst covering the posterior surface of the cord, extending to each lateral side and longitudinally for a distance of about 2.5 cm. The cyst covered the entire eleventh and a portion of the tenth dorsal segments. It was undoubtedly an arachnoidean cyst with probably complete but exceedingly diaphanous walls. The cyst wall was torn through during a little traction. It was opened freely and all the fluid allowed to escape. Unfortunately none of the fluid, which was perfectly clear and had the same appearance as normal cerebrospinal fluid, was collected for chemical and microscopic examinations. The cyst does not belong to the echinococcus or cysticercus class. The cord with its closely adapted pia was now free to sight and palpation, and seemed normal. The dura was closed without drainage. This was followed by a closure of muscles, fascia and skin. A great deal of pain and peculiar vise-like cramps in the legs and lower abdomen were experienced for some days following the operation. With a gradual recession these disappeared in about two weeks. There was retention of urine for eleven days, or about until the patient commenced to sit up, when normal micturition appeared rather quickly.

Third Examinatioin (May 22, 1913).—The patient walked unsupported but somewhat unsteady, showing a gait with an incoordination and mild motor weakness. Left leg was weaker than the right. She states that "the left leg gives way at the hip." Individual tests showed a fair amount of power and movements at hips, knees and ankles, right side being slightly stronger than the left. All deep reflexes were present, right patellar being slightly more pronounced than the left. The Achilles were equal and normal. There were no Babinski or Oppenheim phenomena. For a few days both had been present, the latter disappearing some days earlier. It was interesting to observe from day to day the slow evolution and disappearance of the Babinski phenomenon. The sense of position was fair at the hips and right knee, and impaired at the left knee. A greater amount of impairment was present at the ankles and toes, and rather poorer on the left side where there were 30 per cent. incorrect replies. Deep pressure sense was obtunded slightly in the lower extremities. The tuning fork vibration sense was normal at the right malleoli, feeble at the left malleoli and knees. It was absent at the pelvic prominences and sacrum. Tactile sense showed the same superior line dividing the normal from abnormal, but much more difficult to determine. It was about normal for the right leg and for the left showed a slight obtunding. Pin-pricks corresponded to the tactile sense. She volunteered "it hurts in the right leg," where as before the opera-

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tion it did not. The trophic state was improving. The buttocks, which formerly "felt like sitting on ice," were much better. The lower extremities did not feel so cold. The skin, which formerly was harsh and dry, was now capable of sweating and felt moist.

Fourth Examination.—After being home one month, the patient returned for a brief examination on June 27, 1913. She stated that there had been a continuous improvement in every way. No pain or compression symptoms were being experienced. The urinary functions were normal. She walked rapidly without any support. The gait was not yet quite normal. She readily stood raised on the toes of both feet but not on those of one foot alone. The Achilles reflexes were equal and normal. The patellars were almost normal, the left being slightly brisker than the right. There were no Babinski or Oppenheim phenomena. There remained a little disturbance of sense of position for the ankle and toes, giving about 15 per cent. erroneous replies. All forms of sensation had returned almost to the normal state. Deep sensibility was normal everywhere. There was a mild diminution for epicritic and protopathic sensibility in the left leg, being normal over the abdomen and in the right leg.

No subsequent examinations were made, but reports by letters indicate that the patient was getting along nicely, the last report coming a few weeks ago. She was employed in her household duties. Some subjective symptoms were recited which might be attributed to the climacterium, which is being experienced now.

LITERATURE

While we are delving into past writings bearing on this subject there should be borne in mind the numerous possible errors, as reported cysts which are really nothing more than an excess of cerebrospinal fluid in the subarachnoidean space, cysts extending from within the cord or from without caused by some other disease, or the cystic degenerations of tumors of the leptomeninges. All of the earliest cases coming to our attention under captions of "Arachnoid Cysts," "Cysts from the Cavity of the Arachnoid," etc., are complications in which there is greatly increased cerebrospinal fluid in the subarachnoidean cavity secondary to cerebrospinal syphilis, paresis, tuberculosis and numerous other pathologic states. I name a few of those which I have analyzed as follows:

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Quain,[1] 1855; Ware,[2] 1858; Calmeil,[3] 1858; Wilks,[4] 1865; Sutherland,[5] 1871; Browne,[6] 1875.

Bellencontre[7] has furnished us with an early worthy contribution bearing on the subject under consideration. Most of his cases were cysticercus or echinococcus cysts. One, however, might have been a true single primary cyst belonging to our group. This one is a cyst of Professor Charcot, also reported in his work.[8] It was described as a "hydatid cyst" between the layers of the visceral arachnoid and pia in the cervical region, producing compression symptoms.

A few cases of cystic degeneration of genuine neoplasms of the inner meninges are on record. The Leyden case reported by Lenz[9] was examined pathologically by Recklinghausen and a diagnosis of "cystic sarcoma of the arachnoid with internal hemorrhage" was made. There were two cystic cavities in the tumor resting between the arachnoid layers and pia. It contained spindle cells and large ganglion-like cells. The case of Taube[10] is a lymphangioma with cystic degeneration. The origin was from the pial layers. The case of Potts[11] which was demonstrated at operation had a pathologic diagnosis of a softening or hemorrhagic cyst of the inner meninges. It contained a

1. Quain: Large Cyst from the Cavity of the Arachnoid, Tr. Path. Soc., London, 1855, vi, 8.
2. Ware, C. E.: Cysts from the Cavity of the Arachnoid, Boston Med. and Surg. Jour., 1858-9, lix, 259.
3. Calmeil: Deux observations des kystes de la grande cavité de l'arachnoide, Gaz. d. hôp., 1859, xxxii, 514.
4. Wilks: Cyst in the Cavity of the Arachnoid, Tr. Path. Soc., London, 1865, xvi, 6.
5. Sutherland, H.: Arachnoid Cysts, West Riding Lunatic Asylum Rep., London, 1871, i, 218.
6. Browne: Arachnoid Cysts, Jour. Psych. Med., London, 1875, n. s., i, 167.
7. Bellencontre, P. E. D.: Contribution à l'étude des kystes hydatiques comprimant la moelle épinière, Paris, 1876.
8. Charcot: Lectures sur les maladies du système nerveux, Paris, 1873.
9. Lenz, G.: Beitrag zur Kenntnis der Tumoren der Pia und Arachnoidea spinalis und des Ruckenmarks, Bern, 1895.
10. Taube, J. L.: Lymphangiom der Pia Spinalis; Druckmyelitis, Neurol. Centralbl., 1887, vi, 247.
11. Potts, C. S.: Intradural Cysts of Spinal Meninges Removed by Operation, Jour. Nerv. and Mental Dis., 1910, xxxvii, 621.

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soft gelatinous mass and lay posteriorly over the first lumbar, twelfth and eleventh dorsal segments. It does not belong to this group of simple cysts.

H. Schlesinger[12] has been quoted in a discussion of this subject. He has covered his material in an elegant and thorough manner. The two cases which he has been given the credit of describing as simple cysts of the inner meninges, however, are not clearly cysts of this class. He discusses a number of parasitic cysts.

Oppenheim[13] writes of a local arachnitis above a tumor. The two cases which he has placed on record are questioned even by the author. He considers the possibility of some neighboring tuberculous disease.

Under "Chronic Spinal Meningitis," Horsley[14] has described a condition with allusions to twenty operative cases. Only one of these reported cases is to be considered as a possible simple arachnoidean cyst. Gowers referred this paraplegic for a laminectomy. A recovery followed.

F. Krause[15] in his surgical text uses the term "arachnitis adhaesiva circumscripta," and quotes to some length the historical details of two of Oppenheim's cases. There is a question about the simplicity of both cases, Krause believing the cord changes of at least one to be secondary to compression from the arachnoidean accumulation of fluid.

While Munro[16] reports having seen the pathologic process a number of times, it is necessary to reject his reported cases. They are not true arachnoidean cysts.

The French literature contains almost nothing relating to the subject. I have had recently some personal

12. Schlesinger, H.: Rückenmarks- und Wirbeltumoren, Vienna, 1898.
13. Oppenheim, H.: Geschwülste in Bereich des Centralen Nervensystems, Berlin, 1907.
14. Horsley: Chronic Spinal Meningitis, Brit. Med. Jour., Feb. 27, 1909.
15. Krause, F.: Chirurgie d. Gehirns und Rückenmarks, Berlin, 1911, ii, 726 and 729.
16. Munro: Circumscribed Serous Meningitis of the Cord, Surg., Gynec. and Obst., March, 1910, p. 235.

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conversations on arachnoidean cysts with neurological men in Paris. They informed me that primary cysts of the arachnoid probably are encountered in other countries but not in France.

In reviewing the case of Claude and Rose,[17] it has occurred to my mind that possibly they were dealing with a cyst of the inner meninges, either a complete one or a well-defined pouch.

Of the few undoubted cases of arachnoidean cysts available in the literature, that of Spiller, Musser and Martin[18] holds an eminent position. It is the first concise, definite presentation of the subject. The distribution of the pain rather than motor or sensory changes led to a laminectomy exposing the third and fourth lumbar segments. A very thin-walled cyst of the arachnoid was opened. A gradual improvement covering months ensued. A report[19] six and a half years later indicates an almost perfect recovery, some slight buttock pains remaining.

Mendel and Adler[20] report one undoubted case with an operation. Eight months following the operation the patient was able to walk, though he had not been able to stand on his feet before the treatment. There remained only a slight weakness of the right leg. The arachnoidean cyst was located at the upper dorsal segments.

The case of Bliss,[21] diagnosed as a pia-arachnoid cyst and demonstrated by a laminectomy, seems definitely to have been an arachnoidean cyst with fluid like the cerebrospinal. It compressed the lower cervical segments. Seven weeks following the operation there was in progress a steady return from the paraplegic state to the normal.

17. Claude, H., and Rose, F.: Kompressionssymptome am Rückenmark bei einer Hysterischen, Neurol. Centralbl., 1908, xxvii, 889.
18. Spiller, Musser and Martin: Cysts of the Spinal Cord, Univ. Penn. Med. Bull., March and April, 1903, pp. 27 and 56.
19. Spiller: Am. Jour. Med. Sc., January, 1909, p. 95.
20. Mendel and Adler: Zur Kenntnis der Meningitis serosa spinalis, Berl. klin. Wchnschr., 1908, xlv, 1596.
21. Bliss, M. A.: Cysts of the Spinal Canal, The Journal A. M. A., March 13, 1909, p. 885.

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Bruns[22] has placed on record a certain case. By the highest zone of sensory disturbance, reaching to the sixth dorsal segment on the right and seventh on the left, the lesion was located beneath the fourth dorsal spine. A typical arachnoidean cyst was exposed by a laminectomy at the third, fourth and fifth dorsal vertebrae. The paraplegic state grew worse for two months, which was followed by a period of improvement sufficient to permit walking in nine months.

To recapitulate then, we have but four previously reported cases that might be classed with absolute certainty as primary, simple, uncomplicated cases of leptomeningeal cysts. With my two added cases there is a total of six, and all have been located in the arachnoid.

PATHOLOGY

To appreciate fully the mode of formation of these cord arachnoidean cysts, the neomembranes, and their relationship to important structures, it is first necessary to have a clear idea of the normal gross anatomy and histology of the three coverings of the spinal cord with their relationship to the spaces and cord itself.

Beneath the heavy fibrous dura is the subdural space containing only a minute quantity of fluid. The inner surface of the dura has an endothelial lining. Going toward the cord there follows a thin, delicate arachnoid. This membrane has on the parietal and visceral surfaces an endothelial lining. These delicate cells have various forms, but chiefly ovoid, and all have the same kind of nuclei. Between the endothelial layers are the fine and some coarser connective tissue fibrils. The fibers never form bundles. Mattauschek[23] has demonstrated delicate "epithelial" cells on some of the fibers. Obersteiner[24] teaches that in the normal state the arachnoid is cloudy only in extremely old

22. Bruns, L.: Ein Fall von arachnoidaler Zystenbildung, Neurol. Centralbl., Leipzig, 1911, xxx, 1026.
23. Mattauschek, Emil: Ein Beitrag zur Kenntnis der Arachnoidea spinalis, Arb. a. d. Neurol. Inst. d. Wiener Univ., 1908, xvii, 150.
24. Obersteiner: Nervösen Centralorgane, Ed. 4, Vienna.

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individuals. The subarachnoidean space is the container for the circulating cerebrospinal fluid. Some fibers are stretched across this space, attached from the inner arachnoid to the outer pia mater. There are no nerves or blood vessels in the arachnoid. The pia, adapting itself closely to the cord and its indentures, has an abundance of vessels and nerves.

Key and Retzius[25] have furnished us with some early information on the anatomy of the leptomeninges. Brunet[26] with C. Robin has commented on neomembranes forming in the arachnoid, giving hemorrhages as the cause. Knox[27] furnished a very early argumentative discourse, criticizing Magendie, Sharpey and others. He mentions fine bands connecting arachnoid and pia. He also demonstrated a space completely encircling the cord.

The nature of the disease has prevented satisfactory studies of the pathologic anatomy in the few cases reported. The exceedingly delicate walls do not readily permit the operative excision of even a portion for microscopic preparations. No partitions of the cavity have been observed in the six cases. The contained fluid seemed to have been the same in all and not very different from cerebrospinal fluid in the free subarachnoidean space.

In the etiologic survey it is interesting to note that the six cases were all in women, and that the ages ranged from 17 to 50. A number of theories have been advanced regarding the cause. The parasites can be excluded. Horsley suggests the gonococcus as a cause, which is a possibility. The influenza bacillus might be considered. In fact, any one of a large number of infectious organisms may be the cause. Trauma is another possible factor. Likewise develop-

25. Key und Retzius: Studien in der Anatomie des Nervensystems, Stockholm, 1875, i.

26. Brunet, D.: Recherches sur les néomembranes et les kystes de l'arachnoide, Paris, 1859.

27. Knox, Robert: Some Remarks on the Structure and Arrangement of the Spinal Arachnoid, Lond. Med. Gaz., 1843, ii, 463.

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mental malformations in the arachnoid should be considered seriously. I believe that there may be several provocative agents in the different cases and that from one of the irritating factors there are formed adhesions with the endothelial cells and fibers of the arachnoid. This is followed by an exudate or secretion from the delicate endothelial cells lining the fibrils or covering the interior of the cyst. Thus there is a very slow accumulation of fluid followed by the gradual development of the cord compression symptoms.

DIAGNOSIS

Time does not permit an exhaustive review of all the possible symptoms or the differential diagnosis in cysts of the leptomeninges of the cord. My two cases combined illustrate about all the symptoms observed. The diagnosis of a compression of the cord should usually be made without much difficulty by an experienced diagnostician in neurologic diseases. It is essential that we have an intimate knowledge of the minute anatomy and physiology of the spinal cord and roots. The almost exact metamerism of the spinal cord and its utilization should be of signal service in determining the location of the level of the lesion. To determine whether the lesion is intramedullary, intradural or extradural is not so easy a task. Much importance is attached to the painful symptoms. An intramedullary growth or cyst gives no pain unless indirectly pressing on roots. A carefully prepared sensory chart is absolutely essential, and is the chief guide to the segmental localization. The reflexes are of some importance for locating the lesion. A chronologic review of the development of the motor weakness and the various painful neural phenomena is of much importance in determining whether the pressing object is a hard, soft or cystic one. Variability in the degree of some of the symptoms, especially pain, sensory disturbances and motor weakness, is characteristic of an extramedullary cystic compression.

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Rachiocentesis is advised in all these cases. It aids in establishing the nature of the trouble. Gendron[28] suggests a puncture above and below (*au-dessus et au-dessous*) the compression for separate chemical and microscopic examinations, as has been done by Sicard and Foix in a series of cord compressions from Pott's disease.

TREATMENT

More than a quarter of a century has elapsed since the first successful removal of a meningeal tumor compressing the cord was accomplished by the late Sir William Gowers[29] and Horsley. To-day the spinal canal is so frequently opened that it is unnecessary to say anything regarding the comparative safety with which these major operations may be performed.

Laminectomy is the only treatment for cysts of the cord arachnoid and pia. It is usually necessary to remove at least three laminae. The leptomeningeal cysts have been found chiefly in the dorsal vertebral canal. No postoperative drainage is necessary. No physical or chemical therapeutics are considered for the eradication of the disease. If there has been much motor impairment, however, it might be of considerable benefit to give massage and electrical treatments to the paraplegic regions during a certain period following the operation. The prognosis is excellent if proper treatment is given early. There should be in the neighborhood of a 100 per cent. recovery rate if an early operation is performed. Comparing my two cases illustrates this fact. The compression may continue to a degree at which the pyramidal tracts have degenerated beyond repair, and a restoration of the paraplegic state becomes impossible. My second patient had visited a famous surgical clinic a week or two before coming under my observation, was observed ten days and with grave doubts about the

28. Gendron, André: étude clinique des tumeurs de la moelle et des méninges spinales, Paris, 1913, No. 229.

29. Gowers: Diseases of the Nervous System, Ed. 3, i, p. 627; Med. Chir. Tr., 1888, lxxi.

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diagnosis sent home with the advice to return in three months for further observation after the disease had advanced further. A skilled neurologist should have recognized the condition at a stage fully as early as the patient presented herself.

CONCLUSIONS

1. Admitting, from results of a thorough search of the literature, that there have been recognized only a very few cases of simple, uncomplicated cysts of the spinal arachnoid and pia, we should keep, nevertheless, a lookout for others. The recognition of some of these cysts may have been neglected in the past.

2. The diagnosis of a lesion at or near a certain segmental level should not be difficult. The perception of the exact nature of the compressing material is accomplished less readily.

3. A prompt evacuation of the cystic contents by means of a laminectomy should permit a good prognosis to be fulfilled.

NOTE.—Since the preparation of this article there has appeared the careful report of another case of arachnoidean cyst by Bouché (Compression medullaire par arachnoidite cloisonnée, Revue Neurologique, July 30, 1914, No. 14), the publication being delayed almost one year owing to the bellicose times. The several severely damaged functions had returned almost to the normal twelve months after evacuating the cyst by a laminectomy. The author comments on the dark etiology and the importance of early operative intervention.

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SALVARSANIZED SERUM IN SYPHILITIC NERVOUS DISEASE

SOME SALIENT FACTS: THE METHOD, THE DRUG, THE RESULTS

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The clinical history of syphilis with its therapy is in the process of making, and is greatly in need of restating. Especially is this true of nervous syphilis. The reports of many investigators are so incomplete, so lacking in scientific accuracy, as to cloud their findings and give rise to great confusion and difference of opinion. Paucity of experience, the failure to follow lines of procedure marked out by previous well-known and competent investigators, substituting therefor personal and theoretical modifications, are all important factors in the production of this deplorable condition. There are but few Noguchis or Marinescos, few Swifts and Ellises.

There must be mutual endeavor; a cooperation of effort, so to speak, along definite lines among those interested in the therapy of syphilitic nervous diseases, if order is to come out of chaos and definite and reliable data be obtained.

Mattauschek found from the examination of 4,134 cases of syphilis that 3.19 per cent. of the patients infected suffered from cerebrospinal syphilis. If we believe with Robertson and Klaudner that syphilis is a septicemia, that the organism is invaded by the spirochetes within a day or so after infection, that the nervous system is involved from the beginning, and that the majority of syphilitics are essentially candidates for nervous syphilis from the very outset, the all-important thing in the treatment of this disease is the

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early recognition of the primary lesion. This is readily effected by the means of dark-ground illumination or a microscopic examination of the exudate of the sore, be its resemblance to a hunterian chancre never so slight. This is the psychologic moment in the treatment of syphilis.

Gibbard and Harrison have observed that relapses are more than doubled when medication is begun in the secondary rather than the primary stage, and they believe that by the employment of modern therapeutic methods syphilis may be limited to its earliest manifestations and that eventually congenital syphilis will cease to exist. A most valuable addition to our knowledge of syphilis is the significant fact that the nervous system is subject to an early spirochetal invasion. Serology has enlightened us greatly on this point. Ravaut has found an abnormal spinal fluid in 67 per cent. of his cases of secondary syphilis; Dreyfus in 80 per cent.; Robertson and Klaudner in 70 per cent.; Gennerich in 90 per cent.; Mattauschek in 80 per cent.; Nonne and Mantoux in 40 per cent.; Wile and Stokes in from 60 to 70 per cent., and the latter believe that in every case of secondary syphilis there is more or less involvement of the nervous system. Wechselmann states that in the majority of cases of syphilis the spinal fluid shows an increased protein content. An examination of the spinal fluid of 221 patients in the early stages of syphilis by Wechselmann, Wilhelm and Dinkelacker, showed positive findings in 158, and of these there were nervous symptoms in 98; in the 63 negative cases there were nervous symptoms in 32.

Similar investigations of the primary period have elicited equally interesting results. A pathologic spinal fluid is common; an increase in the albumin and globulin content, with or without pleocytosis, is often observed. The Wassermann reaction is the least to be relied on; a negative spinal fluid is of diagnostic value only when all symptoms indicative of nervous involvement are absolutely lacking. One of Krida's

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patients, with a chancre on the lips of two weeks' duration, showed a marked increased protein content in the spinal fluid.

Certain neurologic findings, such as headache, involvement of the second and eighth nerves (the latter, according to Benario, being more frequently involved in early syphilis than any of the other cranial nerves), modification of sensibility, changes in the reflexes, positive Babinski, pupils unequal and differing in their reaction, etc., are frequently demonstrable in both stages.

"The way the central nervous system is involved in early syphilis," says Coriat, "is certainly surprising, and this is also borne out by the spinal fluid tests. I am making complete neurologic examinations of all cases of constitutional syphilis, in all of its stages, without nervous complaints." [1]

The neurologic findings may, or may not, be associated with definite serologic changes. Either one of these may exist independently of the other. Quincke and Mayer consider that there is a parallelism between pleocytosis and the neurologic symptoms; the former is believed to bear a relation to the severe headaches (Jeanselme and Barbe). If the serologic changes only are present we have what Ravaut calls the "preclinical" stage of nervous syphilis, which may last for months or years before the first clinical sign makes its appearance. I have now under observation two cases in which fifteen years elapsed between infection and the first manifestation of nervous symptoms. Serology enables us to recognize this preclinical period in its incipiency when treatment is most effective.

THE METHOD

As to the best method for the use of salvarsan in nervous syphilis, the profession is hopelessly at variance. There have been too many contributions by

1. Dr. Coriat's work along this line will appear when he and Dr. Sanborn publish their results of the treatment of nervous syphilis based on over 700 injections.

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men whose training and acquaintanceship with the more delicate methods of diagnosis were too faulty to permit of their drawing reliable neurologic conclusions. "The stage of temperate and critical criticisms of results," says Schwab, "has evidently not yet been reached." There are two modes of treatment that merit serious consideration, viz., the intensive and the intraspinal methods. The former, when given intravenously and combined with the intramuscular injections of mercury, is of unquestioned value; some believe that the results are in every way equal to those obtained by the intraspinal methods (Sachs, Strauss and Kaliski). This procedure, when practiced by the injection of salvarsan at the upper part of the nates, in the loose tissues between the subcutaneous fat and fascia (inunctions or injections of mercury being given in the intervals), may be harmless and practically painless, but in the light of the studies of Tilney and Woolsey in vital staining with solutions of trypan blue, is clearly inefficient. These investigators have shown that if solutions of this agent are injected subcutaneously or intra-arterially, the skin and thoracic and abdominal viscera stain readily while the central nervous system absolutely escapes. If the solution be injected intravenously, the stain reaches the dura and pia mater, but does not penetrate the nervous tissue. When the injection is given intraspinally, the membranes are affected as before and the dye penetrates the nervous tissues through the walls of the veins and capillaries, affecting even the epithelium of the latter. These experiments would indicate that intravenous injections of salvarsan are not sufficient in parenchymatous syphilis, however valuable they may be in luetic meningitis, endarteritis and gumma. It has also been demonstrated that diseases of the meninges and their adjacent nervous tissues are best therapeutically influenced by the employment of intraspinous methods. If, as Mott states, the cerebrospinal fluid is the lymph of the nervous system (and it has never been positively

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shown that it does not reach the most remote nervous tissues by way of the subarachnoid spaces and their communicating canalicular system), it is by this medium and through its channels that therapeutic agents can best be conveyed to the lesions deeply situated in the cortex.

Since affections of the meninges, such as influenzal, pneumococcic, and epidemic cerebrospinal meningitis, are not responsive to any other than subarachnoid therapy, it would seem to follow that syphilitic affections of the membranes and their adjacent nervous tissues can best be treated by the intraspinal administration of spirocheticidal drugs.

Intraspinal medication is one of the most notable of the many notable achievements of modern medicine. The intraspinal method, like every new procedure, has suffered more at the hands of its overenthusiastic friends than from the most pitiless criticism of its enemies. The conclusions of partisanship are based on the illusions of hope, not on the solid foundations of clinical and biologic fact. I can best make clear the present status of intraspinal medication by a brief review of its literature and by a few excerpts from personal communications to me from representative American workers.

Ehrlich, in his address on chemotherapy before the International Medical Congress, August, 1913, referred to the intraspinal use of salvarsanized serum as practiced at the Rockefeller Institute, as being "not only novel but suitable in so far as it obviates all possible ill effect on the sensitive central nervous system, by employing a serum obtained from the patient himself, while at the same time it is possible to apply the curative agent in sufficient quantities."

Drs. Purves Stewart and Harry Campbell both advise the treatment of nervous syphilis by intraspinal injections. "It is too early to claim," says the former, "that we possess a cure for tabes or general paralysis, but we can, in suitable cases, do a good deal toward

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arresting both diseases; time alone can tell whether such arrests are permanent." "In parenchymatous syphilis," says the latter, "the intrathecal treatment is the only one yet devised which is capable of arresting the morbid process."

Mott, in his address before the section of Neurology and Psychological Medicine of the British Medical Association at its last annual meeting, said that "the late degenerative forms of syphilis of the nervous system (and I refer especially to general paralysis) have not been cured nor even greatly benefited by any treatment with salvarsan or neosalvarsan, whether administered intravenously or intrathecally." He admitted, however, that his personal experience did not warrant his speaking authoritatively. In the ensuing discussion McDonagh stated that he had observed some remarkable results from the intraspinal injection of salvarsanized serum in cerebrospinal syphilis, but no better than he had obtained from inunctions of mercury and intravenous injections.

Turner stated that it was too soon to express an opinion, but it seemed to him that as good results were obtainable from inunctions as from intraspinal injections. Grainger Stewart believed that in nondegenerative nervous syphilis, intraspinal injections undoubtedly accomplish good, while in paresis and tabes their value was not yet clear.

Eskuchen thinks that the Swift and Ellis method does not influence the objective tabetic symptoms, that the subjective symptoms can be benefited and in some instances the disease is apparently arrested. Myerson is hypercritical in his objections to the use of salvarsanized serum; he suggests, however, that further trial be made in selected groups of cases. Krida states that it is imperative to eradicate every manifestation of nervous early syphilis, from both the clinical and the laboratory standpoints, and that it is here, where the usual methods fail, that intraspinal therapy finds its largest field.

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According to Cutting and Mack, the treatment of general paresis with salvarsanized serum "may not be a solution of the problem, but the work thus far shows that it deserves a thorough trial."

"Personally," says Dr. Coriat, "I highly favor the treatment of syphilitic and so-called parasyphilitic diseases of the central nervous system with salvarsanized serum."[2]

Dr. Jelliffe says:

> There is no doubt that the Swift and Ellis method is along the right lines. My opinion is that there is much to be done in the way of perfecting the technic and that at the present time a number of new avenues are opening up, and we cannot really tell what the future is going to be. Certainly there is very little danger, although some bad result have been met with. On the whole, it is a step forward and not backward.

Dr. Sanborn says:

> I cannot state to you the attitude of the Boston men in general in regard to this treatment, but I believe those who have had any number of patients under observation think that the treatment is rational and is productive of much good and is more efficient than intravenous salvarsan alone. I have personally administered 372 intraspinal injections in private cases, and either I myself or my assistants have given a total of 720 of these injections. I have not been successful in rendering the spinal fluid negative in paresis; in tabes a negative Wassermann may be obtained in most cases if a sufficient number of treatments are given.

There are four kinds of intraspinal medication: first, the injection of mercurialized serum, as advocated by Byrnes; second, the procedure of Swift and Ellis—the in vivo method; third, the in vitro method, viz., the direct intradural injection of neosalvarsan as practiced by Wechselmann, Marinesco, Marie and Levaditi, and Ravaut and Wile; fourth, the employment of both the in vivo and in vitro methods as suggested by Cotton and Smith.

Following along the lines of investigation laid down by Horsley and Ravaut, Byrnes satisfied himself that

2. The citations made from Drs. Cotton, Coriat, Jelliffe and Sanborn are from personal communications to the writer.

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the intraspinal injection of mercurial salts was to be preferred to the unstable, complex arsenical preparations, and he selected the albuminate of mercury as being the most suitable for this purpose. He has reported thirty-two cases of nervous syphilis treated in this manner, and he regards it as equal in efficiency, if not superior to, salvarsanized serum. Cotton says that caution should be observed in its administration.

I believe that the clinical and serologic results thus far obtained make good the claim of efficiency for the in vivo method, and I agree with Dr. Smith that the untoward results observed in intraspinal medication are due to "the so-called later improvements and simplifications of technic," and the direct method of injection. Another year's observation but confirms the assertion of Dr. Hammes and myself before this section last June that "the preference of this method [Ravaut's] . . . over the safer and equally effectual Swift and Ellis process is to us unjustifiable and inexplicable." Sachs, Gordon and Weisenburg have during the past year reported deaths following the use of the Ravaut method. Corbus and also Fordyce advise that the Swift and Ellis procedure be the one of choice. Personally, I think the in vitro method is basically wrong; I cannot conceive it possible to inject intradurally any known arsenical compound in sufficient strength to become absolutely spirocheticidal without at the same time compromising the welfare of the patient. The arsenical preparation that will attach itself to the guest and not affect the host is yet to be discovered. Coriat believes that the good effects of salvarsanized serum are due not so much to the arsenic as to the antibodies it liberates. According to Stühmer, it is very difficult to determine whether antibodies play any additional therapeutic rôle; the good effects of the serum, he thinks, are due, not to free salvarsan, but rather to some oxidation products which by heating are separated from their loosely synthetic combination and again become active.

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Cotton advocates a more strenuous therapy in the future than in the past. The small amount of salvarsan given intraspinally every two weeks he regards as effective only for a few days. It does kill some spirochetes; then in the intervening time the progress goes on and the remaining spirochetes continue to be active. During all this period the patient is deteriorating. When finally the biologic reactions become negative the subject is in a mildly demented state from which recovery does not appear probable. He advises a combination of the Swift and Ellis method and the Ogilvie modification, using the former one week and on alternate weeks the latter. Smith has recently reported twelve cases in which he had used the Swift and Ellis with a modified Ravaut method; the latter was followed by unfortunate complications after the second injection, and was discontinued. He now injects as high as 30 c.c. of undiluted serum weekly (Swift and Ellis) with a more speedy response and no ill results.

The true rôle of subdural therapy in nervous syphilis is one of prevention rather than cure. The frequent occurrence of changes in the nervous system and spinal fluid in early syphilis cannot be too strongly emphasized.

Hauptmann says that a positive Wasermann in the cerebrospinal fluid in secondary syphilis is an expression of a true syphilitic cerebrospinal disease. A neurologic and laboratory examination should be made in every case, for only by an early recognition of the nervous invasion with prompt and radical treatment can subsequent inflammatory and degenerative changes be guarded against.

THE DRUG

"Salvarsan," says Wassermann, "is in my opinion the mightiest weapon in medicine." Gibbard and Harrison state that when mercury alone was used there were 83 per cent. of clinical relapses, but with the use of salvarsan and mercury, only 3.9 per cent. had

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recurred. They prefer it to neosalvarsan, as they regard it as much more active in corresponding doses. According to Nelson and Haines, salvarsan has given them in nine months 64 per cent. of negative serums, whereas neosalvarsan gave 33.3 per cent., and they think salvarsan should always be used if one wishes to give his patients the best possible results in the shortest possible time. "Salvarsan is the most potent remedy," say Craig and Collins, "in our armamentarium in the treatment of syphilis of the nervous system." They believe it to be much more potent than neosalvarsan.

Wechselmann thinks that salvarsan alone should be used in the treatment of syphilis, and this conclusion is based on the observation of 45,000 injections of this drug. Ehrlich and Dreyfus prefer old salvarsan to neosalvarsan, but use the latter when a mild, nonirritating action is desired, although frequently the treatment is begun with neosalvarsan (Conzelman). Many medical men advise that mercury be administered in the intervals between the injections. Collins refers to potassium iodid as a "blighting delusion" and to the administration of mercury by the mouth as a "futility."

The relative merits of mercurialized and salvarsanized serum, are still *sub judice*. The cases in which the former have been tried have been too few and the time too short to warrant any expression of opinion. As to salvarsanized serum much can be said. According to Hough: "The researches of Ehrlich and others have shown that salvarsanized serum has a distinct spirochetacidal power, a power greater than that exercised by salvarsan itself" (Siebert). Swift and Ellis believe that "there is definite evidence that this form of treatment has a curative action on the syphilitic process."

In the light of all the evidence the diversity of opinion that still exists regarding the utility of salvarsan is most surprising. This seems to be almost as marked as when Ehrlich in 1909 first announced his discovery of this truly remarkable synthetic substance.

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THE LANGE COLLOIDAL GOLD REACTION

Dr. Hammes and I have studied the colloidal gold reaction[3] in sixty cases. Eleven paretics gave the typical paretic curve, namely, a complete decolorization in the first three or four tubes with some changes in the next four—5555443200. In one paretic with an acute maniacal onset the colloidal gold solution decolorized completely in every dilution up to the tenth tube. In one case of clinical paresis with negative blood and spinal fluid, the colloidal gold reaction was negative; one case of taboparesis gave the curve for paresis. In ten cases of tabes the curve was somewhat variable, seeming to bear a distinct relation to the intensity of the Wassermann reaction.

In two cases of typical clinical tabes all reactions, biologic and colloidal gold, were negative. Five (50 per cent. of our tabetics) with positive blood and spinal fluid gave what might be called a tabetic curve, namely, 3444432000, with slight variations in the intensity. An atypical case, in which the symptoms were most suggestive of an incipient tabes or paresis, reacted in the syphilitic zone. Three tabetics gave the characteristic paretic curve.

The curve in our cerebrospinal syphilis cases, six in number, was such as occurs in the syphilitic zone. In one case of clinical cerebrospinal syphilis with a positive Wassermann in the blood and spinal fluid, the gold solution reacted negatively; in another similar case the serobiologic findings were all negative, while the gold solution reacted in the syphilitic zone. In two cases of tuberculous meningitis and one case of pneumococcic meningitis the curves were similar, namely 0123343210. In one case of pneumococcic meningitis associated with spinal syphilis of three months' duration, the curve was similar to that observed in our cases of acute meningitis.

3. I am greatly indebted to Dr. Archibald Leitch for his painstaking care in the investigation of the Lange colloidal gold reaction.

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Two cases of syphilitic endarteritis and apoplexy with positive blood and spinal fluid gave a typical paretic curve. A case of tabes associated with cerebrospinal syphilis gave an atypical curve suggestive of both. Two cases of spinal cord tumor, one a fibromyxoma in the cervical region, one a cyst in the lumbar region, gave similar curves: 0000001233. We have found in the following conditions no reaction: a case of transverse myelitis, three of multiple sclerosis, five cases of essential epilepsy, one of involutional melancholia, three normal spinal fluids, two cases of Sydenham's chorea, one subacute degeneration of the posterior columns with primary anemia, one Friedreich's ataxia, one progressive muscular dystrophy (Erb), and two alcoholics.

Treatment has produced no change worth recording on the colloidal gold curve except in three cases of tabes.

THE RESULTS

The recent statements of Schwab that "clinical improvement in paresis as a result of this treatment [Swift and Ellis method] is a thing that no one of recognized standing has as yet asserted," and of Ravine that salvarsanized serum administered intraspinally (Swift and Ellis) gives only laboratory improvement; that its chief asset is the original intravenous injection; that clinical recoveries are not reported; that patients have been made decidedly worse, and that fatalities have occurred are in the light of the following facts most amazing.

In speaking of intraspinal therapy (Swift and Ellis) as used in nine cases, Barrett says:

> Enough time has not elapsed to warrant passing final judgment: . . . but the improvement, or at least the increase in the proportionate number and length of remissions, has been encouraging *and justifies the undertaking of this treatment in all early cases.*

Dr. Blumer states that "thirteen cases of general paresis, taboparesis and tabes have been treated with salvarsanized serum *with encouraging results.*"

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Hough, according to Wardner, sums up the work of Swift and Ellis at the Rockefeller Institute, Myerson of the Psychopathic Hospital, Boston, and Asper of Baltimore, as follows:

> There has been marked improvement in the syphilitic inflammatory processes and in many cases the patients with tabes especially have shown pronounced clinical improvement.

Taylor of Boston, in referring to the results obtained by the Swift and Ellis method in the Neurological Department of the Massachusetts General Hospital and in a limited number of private cases, says:

> In otherwise obstinate cases of tabes the results have been encouraging. It is believed that a certain modification in the course of general paralysis may result if taken early. At times it appears that cerebrospinal syphilis yields more promptly to this than to other forms of treatment; several cases suggest the possibility of complete cure; no permanent ill effects thus far have been experienced.

Dr. Hammes and I have administered over 200 injections of salvarsanized serum (the Swift and Ellis procedure). There have been no ill effects, aside from one case of aseptic chemical meningitis in which there was speedy recovery. Our results are very similar to those of Dr. Taylor. Remissions occurred in three out of eight paretics. One patient relapsed after four months; the other two are apparently perfectly normal. The blood serum and cytochemical reactions were practically normal; the spinal fluid only remained mildly positive. Like Dr. Sanborn we have thus far been unable to get a negative spinal fluid in paresis.

In 75 per cent. of our tabetics the spinal fluid has become negative and all but two out of twenty-two cases have been bettered clinically. The gait has improved, the lightning pains have lessened or disappeared, the bladder symptoms have cleared up, while at the same time there has been a decided change physically for the better.

All of our cases of cerebrospinal syphilis have improved clinically, cytochemically and biologically,

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with one exception in which treatment seemed to have no beneficial effects.

Dr. Cotton says that his statistics show that 50 per cent. (paretics) make tremendous improvement and *that includes all cases treated, many of them in the last stages.* Of the favorable cases, and by that he means the new cases, *at least 75 per cent. have shown very marked remissions.* About sixty patients are living outside of the hospital. There have been several relapses in cases which were pretty far advanced and seemed to improve wonderfully at first. He has had no untoward symptoms, but he reports several cases of mechanical meningitis, such as I described a year ago before the American Neurological Association. The patients quickly recovered.

Of the three following cases, one is paresis in which the mental condition became normal after the fifth injection; the other two are tabetic cases in which the improvement clinically and serobiologically is notable.

CASE 1.—B. was first seen Aug. 19, 1914. Insurance agent, 34 years old, family history negative. Personal history: Uses tobacco and alcohol moderately; gonorrhea in 1904; denies syphilis. Present complaint began about June 1, 1914, with lack of mental concentration and inability to handle his business. Gradually became disoriented and confused, had delusions of grandeur, thought he was the best agent in the country, etc.; at times became greatly excited and had to be put in restraint. Physical examination negative. Neurologic examination: Pupils respond sluggishly to light and accommodation; marked increase of knee jerks; otherwise negative. The blood serum and spinal fluid gave a positive Wassermann; the latter showed increased pressure with globulin excess and 12 lymphocytes to the cubic millimeter. The colloidal gold reaction was typical of paresis. He was given eleven injections of salvarsanized serum (Swift and Ellis); his mental condition became normal after the fifth; after the eighth the Wassermann in the blood became negative. When he left us the spinal fluid still reacted positively; its pressure was normal; there were seven lymphocytes to the cubic millimeter and the Noguchi was mildly positive.

I have under my care at present a paretic who was acutely maniacal; after the second injection of salvar-

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sanized serum the excitement disappeared and now he appears perfectly normal, his elation and grandiose ideas have also disappeared.

CASE 2.—C. was seen first June 12, 1914: 29 years old, news agent; family history negative. Personal history negative, with the exception of gonorrhea in 1914; denies syphilis. Present trouble began about one year ago with occasional attacks of shooting pains; for the past three months there has been a tottering gait with difficulty in starting the flow of urine. After a strong laxative the bowels and bladder evacuated involuntarily. For the past three months sexual power has been markedly impaired. During April, 1914, he noticed a feeling of tightness around the waist which has gradually disappeared; he also felt numbness in both feet. Physical examination negative. Neurologic examination: Pupils respond to light and accommodation, but are irregular, the right being larger than the left; marked Romberg; ataxia of both upper extremities; loss of knee and Achilles jerks; impaired muscle sense of both lower extremities; blood and urine normal; blood serum and spinal fluid gave a positive Wassermann. There was increased pressure of the spinal fluid with a positive globulin and 42 lymphocytes to the cubic millimeter. After the third intraspinal injection his blood became negative, and the spinal fluid after the seventh; cytochemically it was also normal. His gait greatly improved, as did also his bladder functioning; the lightning pains have disappeared and he has gained 16 pounds in weight. When I last saw him he was walking without a cane.

CASE 3.—A., 43 years old, married; family history negative; seen first Jan. 11, 1915. Personal history: No alcohol but tobacco in excess. Denies venereal disease, but admits exposure; one child living and well; wife has had no miscarriages. Illness began about twelve years ago with pain and soreness in joints of the feet and both heels, relieved by potassium iodid and inunctions of mercury. These conditions would recur every year or so and were relieved by this treatment. Almost two years ago he noticed that the feet were numb and were awkward and clumsy; about six months ago numbness developed in both hands and up to the elbows, also around the scrotum. Bladder and sexual functions normal. Occasional lancinating pains. Physical examination negative. Neurologic examination: Light response sluggish in both eyes, accommodation normal, left pupil larger than right; marked Romberg, no ataxia of upper extremities and deep reflexes normal. Lower extremities: knee and ankle jerks lost, superficial reflexes normal, impaired tactile sense extending from feet to knees with loss of muscle sense. Blood and urine normal. The blood

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serum and spinal fluid gave a positive Wassermann, with increased pressure in the spinal fluid, Noguchi positive, 18 lymphocytes per cubic millimeter. After the second injection the Wassermann in the blood became negative; after the fourth injection the Wassermann in the spinal fluid became negative in 0.7 c.c.; Noguchi mildly positive, four lymphocytes per cubic millimeter. He has received five injections of salvarsanized serum intraspinally. There has been a decided improvement in the lightning pains and in the gait; the numbness has left the upper extremities and is very much better in the lower; there is a great improvement in the general health.

Treatment is usually regarded as contraindicated in cases of clinical tabes in which the biologic and cytochemical reactions are negative. Recently Dr. Hammes and I have each had such a case in which the intraspinal use of salvarsanized serum produced very decided improvement. The spinal fluid in my patient reacted positively after the first injection.

While in some patients there is a definite periodicity in the pains following an injection, in others their occurrence is most erratic; sometimes, but rarely, there will be no pain at all, at others they will vary in their manifestation from fifteen minutes to five hours. The cessation of tabetic pain Craig and Collins regard as the single greatest benefit following the use of salvarsan.

CONCLUSIONS

1. Every patient with constitutional syphilis, especially in the early stages, should be examined neurologically and serobiologically, and if there be found any symptoms of nervous involvement or an abnormality of the spinal fluid, intraspinal injections of salvarsanized serum should be given until a clinical and laboratory cure is effected. Bernstein advises this procedure as a prophylactic measure in all cases of syphilis, particularly in the primary and secondary stage.

2. Lange's colloidal gold solution is a valuable aid in diagnosis, possessing a marked corroborative value. Paresis gives a characteristic curve (in over 90 per

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cent. of our cases) so constant, say Grulee and Moody, that "one may venture a diagnosis without other findings, either clinical or laboratory." There is a distinctive tabetic curve which was present in 50 per cent. of our cases. The colloidal gold reaction differentiates clearly between paresis and cerebrospinal syphilis. In tuberculous meningitis the colloidal gold solution reacts markedly. Nonsyphilitic cases sometimes give a vague discoloration which may or may not invade the syphilitic zone, *but which has no special significance.*

3. Intraspinal medication (Swift and Ellis method) is a sane and safe form of therapy, and in the hands of competent men is practically devoid of danger. In cerebrospinal syphilis and tabes, results obtained by conservative workers are most encouraging, and in some cases little less than marvelous. When paresis can be treated in the early, the toxic, stage, before serious degeneration of nervous tissue has ensued, an arrest of the disease may reasonably be expected. Is it too much to hope for a cure?

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ABSTRACT OF DISCUSSION

DR. G. W. ROBINSON, Kansas City, Mo.: Dr. Riggs' results do not differ greatly from mine. Some have condemned this treatment after giving one injection of salvarsanized serum. This is no test. I give my patients from six to twelve injections, giving one weekly. I have used it for eighteen months and have given personally about 300 injections. I have treated about thirty paretics. Four did not improve. About half of them are now at home living normal lives. Of course I do not know how long they will remain well and do not say they are cured. I have had no accidents whatever. I use 20 to 30 c.c. of undiluted serum intraspinously. I have not used salvarsan or neosalvarsan directly in the spine. I think it is a dangerous procedure. I also believe it not safe to treat patients sitting. I formerly gave my treatments to patients sitting, but I now have them lying down while taking the treatment. I have never seen anything to equal this treatment in its results in paresis.

I have not seen any Wassermann reactions made negative after once being positive in paresis. But if the patients act all right after taking the treatment, conduct themselves and affairs with ordinary prudence, I think we have helped them

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whether we have made their serologic condition normal or not. Many persons, how many we do not know, are living normal lives with an abnormal serology. I have seen a case of third nerve palsy with contralateral hemiplegia disappear after three injections of salvarsanized serum. It will certainly stop the pains and visceral crises of tabes more quickly than will any other treatment.

Dr. C. R. Ball, St. Paul: Five years ago I began treatment of cases of paresis with some idea of attaining an improvement in them. I took my impulse from a report of the treatment of such cases made by Donath of Budapest in which he used injections of sodium nucleinate to obtain a febrile reaction, and also a leukocytosis. Since then I have used the tuberculin method of Wagner of Vienna, the so-called intensive treatment, intravenous salvarsan injections in combination with mercury, and the salvarsanized serum intraspinally. So far as the results obtained by these different methods, there does not appear to be a great deal of difference. Remissions may occur after all of them. If my limited experience will permit me to form any conclusion as to the preference in method, I would be inclined to say that the febrile and leukocyte-producing methods along with tonic procedures have, all things considered, given the best results. Looking back over this period of five years and noting that all of the cases in which remissions occurred have again relapsed, some of the patients already dead and all of them better off if they were dead, I am beginning to ask myself if any of these methods of treatment in paresis is really worth while. At best only a temporary respite is obtained in a disease which thus far in our therapeutic endeavor has proved to be absolutely incurable. I was in the East recently and I made a particular effort to inquire at some of the large state institutions what their results were with these newer methods in the treatment of paresis. The invariable answer was, "We are not using them any more. The result obtained does not justify the effort." Nonne in a recent communication says that as yet he has been unable to see that these methods produce results in any way superior to the older methods of treatment. During the past three or four years there has been a perfect furor in the treatment of nervous syphilis, and consequently much confusion and a vast amount of contradictory evidence exists with reference to it. It seems to me that one thing is clear, however, and that is that many of the therapeutists arrive at the conclusions which they intended to prove in the beginning of their articles. In my use of the Swift and Ellis method I have followed the advice of Swift and Ellis, which was that the salvarsanized serum was not to be administered in all cases of cerebrospinal syphilis, but only in selected cases, and in those which did not respond to the other forms of treatment. In such cases I do not feel that

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I have obtained any further advantage, and in some cases former improvement has been lost. I do not believe that salvarsan is being used as much today as it was in the beginning of the so-called salvarsan era.

DR. FRANK R. STARKEY, Philadelphia: I am sure we all appreciate Dr. Riggs' efforts in helping to elucidate this subject, but I am convinced that as time goes on we will hear less and less of salvarsan. Last year at Atlantic City much of the program was given over to salvarsan and the discussion was voluminous; the literature was congested with salvarsan articles, and nearly all of this literature and of these discussions were favorable to salvarsan. We have heard all and more than all of the good results obtained from salvarsan, but we have by no means heard all of the bad results. In Philadelphia recently one of our prominent members read a paper in which he said that he had been administering 0.9 gm. salvarsan every four days, and that he got brilliant results, notwithstanding the well-known fact that it takes from three to four weeks to eliminate a dose of salvarsan. What effect does it have on the kidneys and other structures while in the body and while being eliminated? Time alone will show its destructive action on the organs, including the nervous system. That we are now having more cases of early involvement of the nervous system in syphilis when salvarsan has been employed cannot be denied.

At one of our hospitals there has been a number of deaths from salvarsan which have not been reported; on the other hand I saw a patient with paresis in this institution who was having jacksonian attacks at intervals of about fifteen minutes. Salvarsan was advised but could not be obtained. The next day the man was 100 per cent. better. The day following he was still better, although no salvarsan had been used. The third day salvarsan was employed, and all of the improvement that was obtained was attributed to its use. In 1908 I pointed out the relation of the glands of internal secretion to tabes and other nervous conditions, and ten years earlier the relation of these glands and their secretions to each other and to the sympathetic and central nervous systems. I believe that the more closely we examine our tabes patients for disturbances of the glands of internal secretions, the more frequently we will find them. It is not necessary to have a frank case of myxedema acromegaly or Addison's disease, but the balance of equilibrium that normally exists between these glands can easily be demonstrated to be disturbed. The sex glands may be most conspicuously involved, or not infrequently the pituitary or thyroid may be the one showing most prominent symptoms. Pseudomyxedema or hypopituitarism or hypothyroidism may be present secondary to a hyperactivity of these organs. This follows chronic irritation or inflammation

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here just as it does where other organs are involved, and their activity becomes inhibited by destructive inflammatory changes.

DR. G. H. MOODY, San Antonio, Tex.: Many years ago, before we had salvarsan, I saw a good many cases of paresis and we treated them systematically and persistently with mercury. Some of them improved a great deal and were at times in apparently good condition; others did not improve. Since salvarsan has come I have tried very hard to convince myself that we have a remedy for paresis. I have not given it in a very large series of cases (only about forty) but I am sorry to say that my experience has been disappointing. I do not believe I can say for certain that we have cured a case of paresis. I have seen a great many cases improve. I have had many cases referred to me which had been diagnosed as paresis that I did not believe were paresis; those patients have improved, and some are apparently well. I would say also that in the absence of something better I am still giving our patients salvarsan and salvarsanized serum. As to the bad effects which Dr. Starkey mentioned, I cannot say that all of my cases have been free from bad effects. I have had no immediate bad effects, and for that reason I do not believe the technic has been faulty. One of my cases, within a month after the administration of salvarsanized serum, began to degenerate more rapidly than a case of paresis ought to; another in two months, and another in three or four months, degenerated more rapidly than I had ever seen cases do before. Whether the salvarsanized serum had anything to do with it, I do not know; but it points to the same opinion as expressed by Dr. Ball and Dr. Starkey that the possible degenerative effects may have to be reckoned with. We have to remember that patients improved under mercury and these cases apparently stayed better for a long time, and we can at least hope for the same thing from salvarsanized serum; but I am afraid we are going to be disappointed if we promise a cure in paresis or in tabes.

DR. W. J. G. DAWSON, Eldridge, Calif.: Nothing has been said on heredity; the cases I wish to speak about are the feebleminded. After about 800 examinations by the Wassermann test, the report showed that only 4 per cent. or 5 per cent. of our cases reacted. We have given salvarsan to most of them. We began by using the old salvarsan, but in one case there seemed to be a bad result. It did not occur immediately; it was probably a couple of weeks or more before symptoms of meningitis developed. We have been using neosalvarsan in the majority of our cases since and all I can report about it is that it sometimes changes a + + + positive to a negative. However, I can see little or no improvement in the mental condition.

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DR. A. W. HOISHOLT, Napa, Calif.: The latest writers on cerebrospinal syphilis and paresis tell us it is practically impossible to differentiate clinically between the two in many cases; not even at necropsy can one be sure of the difference. That being the case, I think it may account for the variation in reports concerning the cure of paresis. I remember very well (having been about a quarter of a century connected with the treatment of paresis) the energetic treatment that was given with mercury and iodid years ago, and I once reported sixty-nine cases. In many cases I have seen retrogression in their symptoms, and it occurred to me that the fatal terminations were hastened the more energetic the treatment given. That used to be the report of the majority of writers. In regard to salvarsan, I can only corroborate Dr. Ball's experiences. I wish to mention a case which came to me recently which throws a rather peculiar light on salvarsan. The patient was a colleague who had tabes. He had been in the hands of the best men in the country and came to me for an opinion as to whether or not he should have the Swift-Ellis treatment. He had received thirty-two treatments of salvarsan, most of them (twenty-six out of the thirty-two) intravenous injections, and he still survives. I think the old German saying applies to this case:—*"Allzu viel ist ungesund."* I advised him to take a rest from all treatment for a while.

DR. GEORGE P. SANBORN, Boston: Since January, 1913, I have had under treatment by the Swift-Ellis method between 150 and 160 patients with neurosyphilis, to whom nearly 900 treatments have been given. There have been no deaths and no injurious effects so far as I am aware. Salvarsan and salvarsanized serum we have considered theoretically indicated as our best spirocheticides, applied, so far as possible, where they are needed. Treatment is directed to eradication of infection and has no regenerative action on degenerated nerve tissues. Freed or partially freed from the stress of disease, the patient may be expected to improve to the extent his physiologic mechanism is capable of. The efficiency of the method can be judged to a great extent by its effect in causing to disappear the positive Wassermann reaction, pleocytosis, globulin, and possibly the colloidal gold reaction as found in syphilitic spinal fluid. Their disappearance after treatment must be taken as evidence of initial success against the infectious process.

In our group of cases complete serologic examination has been made of all spinal fluids as obtained, so that in each case we have a series of data from beginning to end of treatment. Since pleocytosis may be taken as indicative of syphilitic irritation, its reduction following treatment favors the conclusion that the latter has to some degree controlled the irritative process. In 104 of the cases treated there was initial pleocytosis. After but one combined treatment, the

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average drop in cell count was 40 per cent.; seventy-six patients were treated sufficiently to reduce the cell count to normal. Of these seventy-six, one third became normal after one or two treatments; 56 per cent. after from three to five; 8 per cent. required six, and 3 per cent. more than six treatments. The greatest number of treatments necessary to reduce cell count to normal was ten, in one case.

Globulin in excess is also a sign of syphilitic irritation. In about 20 per cent. of the cases it became negative and in nearly all the remaining, less strongly positive. The Wassermann reaction, always more difficult to reduce than the others, became negative in fifteen of forty-nine tabetics having initial positive reaction. Nearly all the others have shown consistent tendency to become more weakly positive. In no case of paresis has it been possible to secure a negative Wassermann reaction in the spinal fluid.

The colloidal gold reaction, another evidence of abnormal protein, has been carried out as routine and frequently has tended to become less intense as treatment has progressed. Clinical improvement sooner or later is to be observed in a large percentage of cases. Perhaps the best clinical evidence of efficiency of the Swift-Ellis method is the almost invariable subsidence of root pains in tabes, even in cases in which they have been most severe. Clinical expectancy must be tempered by the fact that the treatment can have no direct regenerative action on degenerated tissues.

Dr. H. G. Brainerd, Los Angeles: I simply want to ask Dr. Riggs if he finds any difficulty in keeping the spinal fluid free from Wassermann reaction, cell count and globulin after he has once secured such a thing. We found that it could be accomplished and hoped that our patients were cured, but were disappointed to find that the clinical symptoms recurred and the spinal fluid Wassermann became more positive, with a larger cell count than ever before, and increased globulin content.

Dr. Cecil E. Reynolds, Los Angeles: I wish to mention two cases in further illustration of Dr. Ball's observations:

One was a child about 3 years old who had congenital syphilis—a case of Dr. Ross Moore's. There was no definite paralysis, although the reflexes were altered on one side, but the child was not getting along well and Dr. Moore asked me to give it salvarsan in his absence, which I did—a very small dose. There followed a marked brachial and facial palsy, with deviation of the head to the right, coming on in about two weeks. I turned back an osteoplastic flap and found an arachnoid cyst over the midfrontal and midprecentral areas.

Another case was that of a very athletic young man of 35 who had contracted syphilis eighteen months before. He

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had started a course of intravenous salvarsan in the East and had been continuing in Los Angeles, having had his last dose about six weeks previously. Coming to Los Angeles for another injection, he registered at a hotel in a perfectly sober condition. I was first called to see him in the small hours of the morning. He was quite unconscious and undergoing endless contortions on the floor, having smashed up all the furniture, in a state of acute delirium. I had him taken to a hospital and in spite of all kinds of medication he had fits for thirty-six hours. He regained consciousness once after bleeding, but soon passed into status epilepticus and died after about forty hours. Dr. Stanley Black examined the cortex and could find no cell degeneration, but only acute congestion. I have never before seen such severe epilepsy commencing in such a recent case of syphilis and probably salvarsan had some effect in causing it. In regard to the intraspinal injection of salvarsanized serum, I have been favorably impressed on the whole.

Dr. Francis X. Dercum, Philadelphia: I am convinced that the day has not yet arrived to form definite conclusions as to the value of salvarsan in paresis. It is true that in a number of cases remissions ensue after salvarsan treatment, both intravenous and intraspinous. However, we should bear in mind that remissions equally pronounced and equal in duration have followed the older methods of treatment or simple physiologic procedures, or have often occurred spontaneously. In many instances, too, salvarsan fails to produce any results whatever and there is some reason for thinking that in some instances the intensive treatment shortens the duration of life. We are in grave danger of being carried away by our enthusiasm. During the last thirty or forty years the profession has passed through a number of therapeutic epidemics. I need only recall in this connection the Bergeon treatment in tuberculosis and the hanging treatment in locomotor ataxia, not to speak of the various psychotherapeutic measures which have from time to time been the vogue.

Dr. Philip Hillkowitz, Denver: I cannot speak from the neurologic, but only from the laboratory standpoint, and can only confirm what has been said in the paper and discussion, that as regards paresis we have never succeeded in making the Wassermanns negative. For that matter, in old cases of syphilis of twelve to fifteen years' standing without any cerebrospinal symptoms, it has been impossible to get the Wassermann negative in the serum by specific treatment. As regards tabes, there has been occasionally a return to the negative, but only in a small number of cases. From the theoretical point of view I can only repeat what the chairman has said about the therapeutic efficiency of salvarsanized serum—that by injecting 6 to

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9 gm. of salvarsan and removing a small amount of blood within an hour, the amount of arsenic must necessarily be infinitesimal and we cannot therefore explain *a priori* the supposed beneficial results. If then the clinicians are not so very enthusiastic on this treatment, it stands to reason that we must view the therapeutic effect of this drug with more skepticism than we have done heretofore.

DR. W. A. JONES, Minneapolis: I saw in the National Hospital in London, last year, three tabetics lying side by side. They were the ordinary laborers of the streets, and were more or less robust, more or less alcoholic, and all syphilized. One was treated with salvarsan, one with mercury, the third was given no treatment, and he made much more prompt recovery than did those who were medicated. I wonder if we have not gone to too great extremes and if we had better not get back to more rational lines of observation and treatment. I have had some fatalities in my serum work, and a number of failures and frequent relapses, and on the whole the work is rather unsatisfactory. One fatality was in a most promising case; following his intraspinous injection of salvarsanized serum, he had the most violent attack of purpura hemorrhagica I ever saw and lived only four days after its inception. I think the selection of cases should be very carefully made and the treatment should be very carefully carried out.

DR. WALTER V. BREM, Los Angeles: When Swift and Ellis announced their intraspinous treatment I welcomed it with enthusiasm and used it in about 100 cases; but my enthusiasm soon waned. I have had a number of patients who improved at first. The tests in the spinal fluid reduced considerably with the first and second treatments and then remained there and I could make no further impression on them. I used the whole serum in some cases of this kind and drew the blood fifteen to twenty minutes after the injection of a full sized dose. Swift himself has recently reported that the result of this treatment was probably not due entirely to the salvarsan in the serum; that he had treated two patients with serum alone from syphilitic patients who had not been given salvarsan and had obtained good results both clinically and serologically from the use of this serum.

I was much interested in the mercurialized serum reported by Byrnes, and have given over twenty intraspinal injections of mercurialized serum. I have obtained results in two patients that I could not get with the salvarsan, but in the others I failed. This, however, may be due to the fact that I was unable to use the quantity of mercury Byrnes used on account of the intense reactions, and that nearly all the patients have been paretics. I began by giving one mg. and got a most intense meningeal reaction. This

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patient had 33 per cent. reduction in the Wassermann test of the cerebrospinal fluid after one treatment. The next week I gave him a half milligram of mercury and he had no headache or fever, and had another reduction which made a total reduction of 50 per cent. in his Wassermann test. The next time I gave him another half milligram and found that there had been no further reduction in the spinal fluid. The next time I gave him ¾ milligram and the spinal fluid showed a considerable increase in the Wassermann test. I am unable to give that patient more than ½ to ¾ milligram on account of the intense reaction. The other cases have responded variously, but nearly all have had rather intense reactions.

I believe that the logical treatment of syphilis of the nervous system is by the subdural route, if we can get a drug that will kill the spirochetes without injuring the patient too much. The treatment of epidemic meningitis and of tetanus by the intraspinal use of serums seems to be evidence of that. I think this is the line of work which will eventually become the ideal method of treating these diseases.

Dr. George A. Moleen, Denver: From the diversity of opinion expressed here (which is simply an echo of that which has gone over the medical world) I think one can hardly assume any other position than that of doubt. Beginning with the original statement of Ehrlich, with reference to salvarsan, which certainly led us to jump with enthusiasm; and when Swift and Ellis announced that by the incubation of serum from a previously salvarsanized patient we could by inoculation destroy the spirochete, we also jumped, and the application of it in these cases seemed at that time to be a most wonderful advance.

I was much impressed a few weeks ago when I saw a patient who had been secured against the recurrence of syphilis as a result of a single injection of salvarsan four or five years before in Massachusetts; he showed unmistakable evidences of paresis. On the same day a man of 74 years was referred complaining of headaches and other evidences of arteriosclerosis, and I learned during the taking of the history that he had had syphilis at 19. He had taken mercurial treatment after the secondary flora and had no recurrence whatever, nor had he had further treatment. The comparison of these two cases is to me very significant; and the question arises whether, if we had, or were to have, a syphilitic infection, would we rather have been treated fifty years ago, than now?

The relation of arsenic and the nervous system has been one which heretofore has been looked on with suspicion, because we looked for parenchymatous degeneration following the use of large quantities of arsenic, or even small quantities over a long period of time. This leads up to

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the question as to whether or not these nervous degenerations are the result of the specific virus on tissues and whether the resistance of those tissues is not lowered, and whether it is not even more vulnerable to destruction than it was before. It is a question time alone can decide. It would be interesting, as suggested, to use inactivated, non-salvarsanized serum, such as has been used in epilepsy and in certain forms of skin eruptions, and see if parallel cases taken as control would not show amelioration.

Nucleinate of soda injected in the vein gave good results in paresis and good results in tabes. It may be that the use of inactivated serum might produce parallel results to those we have obtained with salvarsanized serum. I believe we are all more or less in doubt; it requires time, careful observation, and, most of all, conservative judgment.

Dr. C. Eugene Riggs, St. Paul: I am heartily in accord with Dr. Jelliffe that the Swift and Ellis treatment is a step in the right direction, though I am far from feeling that it is the final step. I believe that this form of treatment at present is the most efficient. I was far from making any claim for the cure of paresis; what I did say was that it *increased the tendency to remission.* Before I came here I had under my care a stalwart man in the maniacal stage, so acute that it required ten men to put him in restraint. Three injections of salvarsanized serum had a most marvelous effect. I had a conversation with him before I left home and his talk was perfectly rational.

Dr. Brainerd has asked as to the ultimate effect of this treatment on the serobiologic reactions of the spinal fluid. I am unable to answer this because I have not yet had opportunity to accumulate the necessary data. May I, however, quote a sentence from a letter received recently by Dr. Hammes from Dr. Swift: "The benefit in most of the tabetics seems to have been permanent. In some in whom the Wassermann reaction was not negative in the cerebrospinal fluid, there has been a relapse. But of course we do not consider these patients as cured."

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REFLEX OF THE UPPER EXTREMITY

AN UNDESCRIBED SIGN

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LOS ANGELES

The lack of a sign or reflex in the upper extremity in any way comparable to the knee jerk has always seemed to me to be a real neurologic tragedy. Consequently the search for such a sign has held my interest strongly.

During a poliomyelitis epidemic in Los Angeles five years ago the first idea of the sign which is the subject of this paper was secured. Since then enough material has been studied to warrant this preliminary report.

There is little to be found in the literature bearing on the subject. A German and two French students have studied the tonus in the flexors of the fingers under pathologic conditions. The methods described do not seem to me to be delicate enough to give very much real information. The method now to be described has at least the merit of extreme delicacy to commend it. The estimate of tonus in the flexor muscles of the fingers gained by this method is more precise than that given by any other method of clinical examination known to me. In my experience it makes more possible than heretofore the very early discovery of evidence pointing toward unilateral lesions, either central or peripheral, as for instance a decreased tonus in an oncoming poliomyelitis, or an increased tonus in an arm affected by a developing brain tumor.

TECHNIC FOR STUDYING THE SIGN

First, it will be noted that the position of the normal human hand when at rest includes slight flexion of all

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the joints of all the fingers; the hand is one third or one half clenched. The sign I am describing consists in a study of the muscular tonus responsible for this flexion in the normal hand, together with a comparison of the tonus in pathologic conditions.

The sign is elicited by a sudden extension of the semiflexed fingers, producing an excitation in the muscles. This seems to be quite analogous to that produced by tapping the patellar tendon. Foreign observers have attempted to do this by taking hold of the fingers and giving them a sudden twitch. This method will elicit the more severe hypertonic conditions, such as are found in old hemiplegias, for instance. But it will not give the finer differences observable by my method. The knee jerk is primarily an ocular demonstration, while this sign is to be felt rather than seen.

1. Extend hand to be examined on the forearm enough to take up the "slack" in the finger flexors. Do this by laying your hand flat against the patient's palm and pushing it back to as great an angle as possible with the forearm. If you have placed your hand so that it lies crosswise of your patient's hand you will find that his fingers are curling down over your hand.

2. While still retaining the tension on the flexors in the above-described manner, move the examining hand very sharply along the palm toward and against the curling fingers. Do this several times in quick succession. In this way a very accurate concept of the tonus in the flexors is obtained. Compare the two sides.

A trial of this method on a small number of normal hands will serve as a basis for estimating pathologic tonus. Carrying it out very rapidly time after time will usually overcome the self-consciousness of the patient and his efforts to help in the examination.

Tonus of the flexors of the fingers when tested by this method is found to be decreased uniformly in poliomyelitis involving the upper extremity. This is also true of polyneuritis. In a long series of cases of

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tabes dorsalis examined in the National Hospital in London last summer and in other places this reaction or reflex was found to be absent or much diminished in the majority of cases. A very frequent combination found in this series was absent knee jerks, elbow and wrist jerks present, and flexor tonus diminished.

In central lesions, on the other hand, this sign is uniformly positive. Wherever organic hemiplegias, for instance, show positive reflexes, this reaction is positive. The probable value of the sign lies in the fact that at times it can be demonstrated unusually early in the symptomatology of central lesions; that is, an increased tonus develops. Thus it has been noted in brain tumor when no other localizing signs could be found except diminished abdominals and epigastrics. A true finger flexor clonus has also been found occasionally in upper lesions.

A study of the physiologic questions involved in this sign leads me to conclude that the stimulus given to the flexor muscles by this method of sharp and sudden straightening of the fingers is of the same order and quite comparable to the stimulus caused by tapping the patellar tendon. Certainly it has reacted at all times quite parallel to the knee jerk in my experience. If it can be interpreted as showing in the upper extremity what the knee jerk tells us in the lower, its value will be fairly established. One thing is sure: a little practice with this sign will persuade the careful observer that infinitely more delicate differences in muscular tonus can be detected by this technic than by any of our ordinary methods.

In perfecting this technic it has been the custom to examine for the sign in every case seen. In this way it has been determined that relative strength in the two hands of a normal subject has no influence on the sign. It is also found that the normal tonus is different in different individuals. Relative training of the hands, as pronounced right or left handedness, is without effect on the sign.

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THE DIAGNOSIS OF TUMORS IN THE POSTERIOR CRANIAL FOSSA

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Our knowledge of cerebellar symptoms and of cerebellar localization has advanced considerably during the last few years, chiefly owing to the work of Babinski and Rothmann, but in comparison with *cerebral* symptomatology and localization this progress has not been great. This is mainly because clinicians have not studied their cases with sufficient care, and when diagnosing tumors in the posterior cranial fossa have been satisfied with gross diagnoses.

Recently one of us, with Dr. Mills,[1] in a paper on cerebellar symptomatology and localization, reviewed most of the literature on this subject, both foreign and American. In only a few cases was any attempt made to differentiate cerebellar symptomatology to any of the limbs or parts of the trunk, while a study of the pathologic reports was equally disappointing; most writers were satisfied with the assertion that either one or both lateral lobes or the vermis was implicated, there being few definite statements of the particular part of the cerebellum diseased.

It is advisable, therefore, briefly to state our views on cerebellar localization and cerebellar symptomatology. We have adopted the tentative localization

1. Mills, Charles K., and Weisenburg, Theodore, H.: Cerebellar Symptoms and Cerebellar Localization, Including Kinematographic Observations on Cerebellar Phenomena, THE JOURNAL A. M. A., Nov. 21, 1914, p. 1813.

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advanced by Mills and Weisenburg. It is probable that many of their views are wrong; but it is necessary to make a start, and the localization advanced by them is perhaps as comprehensive as can be made at the present time. To arrive at a definite conclusion it is necessary for all clinical and pathologic reports to be accurate and complete, so as to leave no doubt as to what part of the cerebellum is implicated.

We believe that the chief function of the cerebellum is to synergize all movements of the body. Whatever symptoms are present, such as hypermetry, adiadokocinesis or tremor, result from loss of synergy. This asynergy can be detected in any part, or parts, of the body. It may be present in one limb, the limbs of one side, in the upper limbs and shoulder girdle, in the lower limbs and the pelvic girdle, or in all parts of the body. Not only that, but in the development of these symptoms it is possible to tell whether the lack of synergic movement in the upper limb, for example, is in abduction or adduction, or in upward or downward movement. The methods of elicitation have been carefully described in the paper previously referred to. We wish to emphasize here that it is possible to differentiate cerebellar asynergy in parts of the body.

We have found the Bárány method of investigation of great value in the differentiation and elicitation of symptoms. By means of the caloric tests and the turning tests, it is possible to demonstrate by direct stimulation of the vestibular apparatus the presence or absence of certain phenomena. This subject was presented by one of us in association with Dr. I. H. Jones, before the recent meeting of the American Neurological Association in New York.

In presenting this subject, we first discuss lesions confined strictly to the cerebellum; second, lesions implicating the superior cerebellar peduncle; third, lesions involving the middle cerebellar peduncle; fourth, lesions implicating the inferior cerebellar

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peduncle; fifth, lesions of the cerebellopontile angle; and last, lesions invading the cerebellopontile angle secondarily.

We desire to emphasize that, to make an accurate diagnosis of a cerebellar lesion, it is necessary to take into consideration not only the cerebellar symptoms, but all other symptoms. We have been repeatedly impressed with this fact since our attention has been more especially directed to this subject. Often we have made a diagnosis of a lesion of the labyrinth, only to find the cerebellum implicated, and vice versa; again we have diagnosed cerebellopontile-angle tumors only to find that the angle was secondarily invaded by a lesion growing from the cerebellum. It has not been infrequent to diagnose lesions of the cerebellum when the tumor has been in the third ventricle and has involved the superior cerebellar peduncles secondarily.

DIAGNOSIS OF LESIONS STRICTLY CONFINED TO THE CEREBELLUM

In accordance with our views of cerebellar function, lesions of the cerebellum itself cause more strictly limited symptoms than those which invade any of the peduncles. Most tumors of the cerebellum are gliomatous and are of slow growth. The majority tend to invade the middle rather than the outer portions of the cerebellum, and as a consequence, the vermis is nearly always involved wholly or in part, in addition to one of the lateral lobes. We believe that in the superior vermis are centered the synergic movements of the upper trunk or shoulder girdle and in the inferior vermis the movements of the lower trunk or of the pelvic girdle. As a consequence there will always be in such lesions, in addition to disturbances in the movement of one or both limbs, alterations in the station and gait.

There is a great difference in the station and gait in those cases in which the shoulder or the pelvic

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girdle is implicated. In the shoulder girdle cases, both in standing and in walking the feet are not held widely apart nor is there a swaying of the pelvis. The chief difficulty is that the moment the patient attempts to stand or to walk, his trunk leans or falls forward, backward or to one side very markedly, and *much more so* than in the pelvic girdle cases, for in the latter, because of the inability to synergize the movements of the lower trunk with the legs, the difficulty in walking is much greater, the legs are held farther apart, and instead of the patient lurching to one side, forward or backward, the gait is more irregular, the body is held more erectly, and in walking the legs are projected in front of the patient, showing clearly the lack of synergic action between the trunk and limbs. When the shoulder and the pelvic girdle are both involved, it is sometimes almost impossible for the patient to stand or to walk without aid.

A very good way of differentiating shoulder and pelvic involvement is to place the patient on the floor on the hands and knees. While in such a position, if the pelvis be implicated, there will be very marked to and fro swaying of this part, the shoulders remaining practically still, whereas in the other type the pelvis is held steadily, but the head and shoulders sway and deviate to the side of the lesion.

In cases in which the vermis is implicated, the staggering is mainly forward or backward, rather than to one side, and of such cases, it is more marked in the shoulder girdle type. In our experience, if in addition to the involvement of the vermis, the lateral lobes are implicated, the sway of the trunk is to the side of the lesion, although it must not be forgotten that in a progressive cerebellar disease, the patient may learn, for example, to go voluntarily to the left to offset his staggering to the right.

In those instances in which the lateral lobes alone are implicated, the asynergic movements are present only in the limbs of the involved side: if in the

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superior lobe, in the upper limb, and if in the inferior lobe, in the lower limb. By direct stimulation of the vestibular apparatus it is possible to obtain, for example, a total lack of outward pointing or inward pointing, whereas this is not so readily demonstrated by the ordinary tests.

Nystagmus. — The question whether nystagmus should or should not be present in cerebellar lesions has by no means been settled. According to our views of cerebellar localization we place the synergic center for eye movements in the extreme upper portion of the superior vermis. It is a well-known fact that in chronic lesions strictly confined to the cerebellum, involuntary nystagmus may be present. One very marked case of this type has been carefully studied by means of moving pictures, and was referred to in the paper with Dr. Mills. In this case the nystagmic movements were constant and in all directions—upward, downward, laterally and in a rotatory direction. We regarded these ocular movements as an example of direct cerebellar ocular asynergy. Other examples of nystagmus are found in diseases of the Friedreich's ataxia type. Rarely involuntary nystagmus occurs in progressively developing tumors, but this is not the rule.

If, however, nystagmus be developed by voluntary movement, the lesion is probably extracerebellar. It is especially in these cases that direct stimulation of the vestibular tract demonstrates the true source of such nystagmus. If, for example, the right ear is stimulated by cold water, there will be obtained quick jerking movements of the eyeballs to the opposite side with a slow return to the same side. The slow recoil to the side of the stimulation is the cerebellar movement, while the quick jerk to the opposite side is the cerebral component. The same results will be obtained if the vestibular apparatus be stimulated by means of the turning chair.

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In such tests the impulses go from the labyrinth by the vestibular nerve to the three paracerebellar nuclei — Deiters', von Bechterew's, and triangular nuclei—and thence to the nuclei of the third, fourth, and sixth cranial nerves by the posterior longitudinal bundle, and then to the cerebral cortex. If there be any interruption of this arc, normal nystagmus is not obtained. The location of the lesion will depend on the associated symptoms. If in the eighth nerve there will also be loss of hearing; if in the paracerebellar nuclei, no deafness, but there may be some implication of either the fifth, ninth or tenth cranial nerves, inasmuch as these nuclei are in the outer upper portion of the medulla. If the lesion is in the pons or crus, there may be in addition to the sensory and motor symptoms, involvement of either the seventh, sixth, fifth, fourth or third cranial nerves, with paralysis of associated ocular movement.

Apparently then, the conclusions to be drawn regarding ocular movements are as follows: Involuntary nystagmus is nearly always cerebellar, but voluntary nystagmus is the result of implication of those fibers which are in relation to the vestibular apparatus outside of the cerebellum.

General Symptoms.—The presence of cranial nerve symptoms, as of the seventh, sixth, fifth, ninth, tenth or twelfth, indicates that the lesion is extracerebellar. Dizziness with disturbance of hearing is the result of implication of the vestibular apparatus somewhere in its course and is not a cerebellar symptom. Involvement of the motor fibers, with paresis of the limbs, increased reflexes and Babinski, indicates that there is pressure on the motor apparatus and should not be expected in lesions strictly confined to the cerebellum, except in those cases in which the lesion is very large. As a rule, motor involvement indicates a pontile lesion or a lesion in the angle pressing on the pons. Headache, nausea, vomiting, and choked disk, are, of course, early manifestations, but only

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in those instances in which there is interruption in the flow of the cerebrospinal fluid, and generally indicate a lesion either in the crus or pons pressing on the aqueduct of Sylvius.

LESIONS OF THE CEREBELLAR PEDUNCLES

We possess little definite knowledge of the functions of the fibers in the cerebellar peduncles. It is supposed that the inferior and middle peduncles transmit impulses to the cerebellum, and that the superior peduncle transmits impulses from this organ. It is probable, however, that all the peduncles transmit impulses in both directions. It is also probable that the inferior peduncles are concerned chiefly with synergic movement of the lower limbs, and to a lesser extent of the upper limbs, the middle peduncle with the synergic movement of the trunk and head, and that the superior peduncles are mainly concerned with the association of cerebral with cerebellar functions. All this, however is theory, based somewhat on anatomic and physiologic facts, but not as yet supported by pathologic and clinical data.

In the literature there are a few instances of lesions confined more or less to one of the peduncles, but it cannot be said that our clinical experience is such that we can differentiate the asynergy resulting from lesions of the superior from those of the middle or inferior peduncles. From our present knowledge all we can say is that lesions strictly confined to any of the peduncles cause asynergic symptoms in all parts of the body and as a consequence we are compelled to make the diagnosis not on the cerebellar symptoms but on those associated with them.

There are instances in the literature in which so-called turning or rolling movements have been recorded in tumors of the middle and of the inferior peduncles, either toward or away from the side of the lesion. These cases, however, are not definite and cannot be relied on. There is, however, this differ-

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ence in the cerebellar symptomatology of the different peduncles: Because of the anatomic relation of the superior peduncles, lesions of this part invade mostly both sides, the cerebellar symptoms being present all over the body, although rarely, as in the case reported by Mills[2] in which there was thrombosis of the superior cerebellar artery, the asynergy is limited only to the limbs on one side.

The middle cerebellar peduncles being more widely separated, such lesions are often unilateral, whereas tumors implicating the inferior cerebellar peduncle are almost always one-sided.

Tumors of the Superior Cerebellar Peduncles.—In most instances tumors invading these peduncles grow from the third ventricle through the aqueduct of Sylvius, often bisecting the cerebral peduncle and pressing on or destroying one or both red nuclei. In such cases the symptoms consist of a gradually developing paralysis of associated ocular movement upward, and if the lesion extend laterally, central disturbance of hearing on one or both sides, because of the involvement of the posterior corpora quadrigemina. There may be also partial weakness of both upper and lower limbs with the presence of increased reflexes and the Babinski phenomenon. The symptoms resulting from extension of tumors from the third ventricle into the aqueduct of Sylvius were discussed by one of us.[3] Since then we have seen a number of such cases both clinically and pathologically. Because of the preponderant cerebellar symptoms they are nearly always diagnosed as cerebellar, but with the gradual onset of paralysis of associated ocular movement upward the diagnosis is no longer in doubt.

2. Mills, Charles K.: Preliminary Note on a New Symptom Complex Due to Lesion of the Cerebellum and Cerebello-Rubro-Thalamic System, the Main Symptoms Being Ataxia of the Upper and Lower Extremities of One Side, and on the Other Side Deafness, Paralysis of Emotional Expression in the Face, and Loss of the Senses of Pain, Heat and Cold Over the Entire Half of the Body, Jour. Nerv. and Ment. Dis., February, 1912.

3. Weisenburg, T. H.: Tumors of the Third Ventricle with the Establishment of a Symptom-Complex, Brain, 1910, xxxiii, 236.

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Rarely, as in the case previously referred to,[2] there is thrombosis of the superior cerebellar artery. In such instances there will be loss of unilateral emotional expression in the face on the same side and disturbance of sensation for pain, heat and cold over the other side of the body.

Sometimes large tumors of the thalamus extend downward and implicate the peduncle on one or both sides. In such instances the diagnosis is difficult until the third nucleus becomes involved and with the presence of the paralysis of upward associated ocular movement the diagnosis can be made.

Lesions of the Middle Cerebellar Peduncles.—Tumors in this area are rare. Such lesions usually either extend from the pons into the cerebellum by means of this peduncle or grow from the cerebellum into the pons. Hemorrhages or a thrombosis often give limited symptoms. One such case is now under our observation. This patient, a physician of 45 years, suddenly developed numbness on the entire left side of the body with a complete motor right fifth nerve palsy and a partial weakness of the right sixth nerve. He had besides limited cerebellar disturbances in the limbs of the involved side which were best brought out by the Bárány tests. In this instance the diagnosis was made by the presence of the fifth and sixth nerve palsy on the side of the lesion, the sensory symptoms on the opposite side due to the irritation of the median lemniscus and the slight cerebellar asynergy on the side of the lesion.

The conclusion can be drawn that in lesions of the middle cerebellar peduncles the associated phenomena consist of the fifth or sixth nerve symptoms on the side of the lesion, with sensory and motor phenomena on the opposite side.

Lesions of the Inferior Cerebellar Peduncles.—Such tumors are very rare and we have never seen one ***limited*** to one or both of the inferior peduncles. We

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have, however, seen a number of tumors of the cerebellum in which there has been an extension of the growth into one or both peduncles. In such growths the associating phenomena, if the lesion extends into the medulla, should consist in implication of the vestibular tract, and disease of the ninth, tenth and twelfth cranial nerves.

Lesions of the Cerebellopontile Angle.—The diagnosis of tumors in this area as a rule is not difficult. We have encountered cases, however, in which such diagnosis had been made, only to find that this angle was invaded secondarily by tumors growing from the cerebellum and more rarely from the pons. The differential diagnosis is important from the surgical standpoint, for it is readily seen that tumors growing *from* the pons or cerebellum offer little hope for surgical removal.

Tumors growing in this angle are usually fibromatous and more rarely fibrosarcoma. We have encountered a number of cysts, but these are generally parts of gliomatous tumors growing from the cerebellum. Most tumors grow from the eighth, and more rarely from the seventh, fifth, or sixth cranial nerves, respectively. The first symptoms are usually referred to the cranial nerve from which the tumor grows and in the course of time the seventh, sixth and fifth nerves become implicated. Of course, the symptoms depend on the size of the growth. In small tumors no pressure is exerted on either the pons or cerebellum. In a well-developed case, besides the cranial nerve symptoms, because of the pressure on the pons, there are motor symptoms on the opposite side and more rarely sensory phenomena.

Because of the pressure on the anterior part of the cerebellum, both the superior and inferior surfaces are involved, this causing cerebellar asynergy of the limbs on the same side. In cases in which the angle is invaded secondarily from the cerebellum or from

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the pons, the cerebellar symptoms are always much more marked, and it is because we have not been sufficiently careful in the differentiation of the cerebellar phenomena that errors in diagnosis have arisen.

We wish to emphasize here that in the usual tumor growing from the cerebellopontile angle the cerebellar symptoms ***are not very marked*** and that unless the tumor is very great the asynergy will be limited only to the arm and leg on the side of the growth, and that if there is in addition to the cranial nerve symptoms cerebellar asynergy in the trunk and limbs on both sides, it is probable that the tumor grows either from the cerebellum or more rarely from the pons.

Of course, if the progress of the symptoms is carefully noted, in the latter type of case the cerebellar phenomena should occur first and the cranial nerve symptoms last. But unfortunately we are not always permitted a clear-cut history, and it is because of this that we desire to emphasize this important point in differential diagnoses.

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STUDIES ON THE LOCALIZATION OF CEREBELLAR TUMORS

III. POSTERIOR NEW GROWTHS WITHOUT NYSTAGMUS *

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INTRODUCTION

The purpose of this study has been to determine the proportion of patients with certified subtentorial new growths who have shown no nystagmus previous to operative treatment.

Practically all authorities agree that nystagmus is a valuable localizing sign in diseases of the posterior cranial fossa. To this category belong especially lesions of the cerebellum, the cerebellar peduncles, the vestibular nerves, the neighboring nuclei and the labyrinths. When general pressure symptoms suggest the presence of a tumor, nystagmus assumes more importance still, for it is recognized that the occurrence of this sign in a patient with symptoms of subtentorial new growth argues in favor of an extracerebellar or intracerebellar neoplasm. Stewart and Holmes[1] hold that this sign is almost invariably present in every case of cerebellar tumor at one or another time.

Occasionally, however, in patients of this type no discernible rhythmic movements of the eyes are found on repeated investigation. Though frequently other features of the examination suffice for purposes of diagnosis,[2, 3] there are instances in which the localiza-

* From the Surgical Clinic of the Peter Bent Brigham Hospital, Boston.

1. Stewart and Holmes: Symptomatology of Cerebellar Tumors, Brain, 1904, xxvii, 522.
2. Grey: Studies on the Localization of Cerebellar Tumors: 1. Staggering Gait, Limb Ataxia, The Romberg Test and Adiadococinesia, Jour. Nerv. and Ment. Dis., to be published.
3. Grey: On Localization of Function in the Canine Cerebellum, Jour. Nerv. and Ment. Dis., to be published.

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tion of an intracranial tumor in the posterior fossa is a very difficult problem.

A case in point recently came to my attention (table, 2320). The patient was examined repeatedly and by different observers, but no suggestion of nystagmus ever was noted. The picture as a whole was quite obscure. A tentative diagnosis of subtentorial tumor finally was made from the rapid onset of pressure signs (advanced choked disks, areas of absorption and enlarged venous channels in the roentgenograms, etc.) and the history of some disturbances in gait. There were no further significant localizing signs. Operation disclosed a solitary, median tubercle of the cerebellum measuring approximately 3 cm. in diameter.

When confronted with problems of this sort it is a matter of some importance to know the relative proportion of patients with certified cerebellar tumors who show no nystagmus previous to operative treatment. With information of this nature conclusions may more easily be drawn regarding the advisability of suboccipital exploration.

The records used in this analysis are from patients with tumors in the posterior fossa who have appeared in the neurologic services of Dr. Cushing at the Johns Hopkins Hospital previous to September, 1912, and at the Peter Bent Brigham Hospital since that date. Most of the cases in the latter group were personally studied by me. Of several hundred such cases only those—fifty-one in number—in which the new growths were localized at operation or at necropsy have been used for this report. In addition, for purposes of comparison, the records of forty-one patients from the latter clinic with verified supratentorial tumors have been analyzed for the presence or absence of nystagmus.

Practically all of the patients were studied by more than one observer. This method of checking up the routine examinations is significant, particularly for the patients reported as showing no nystagmus.

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As Wilson and Pike[4] have pointed out, the word "nystagmus," in its present usage, is an indefinite term covering various types of ocular movements arising from lesions in diverse parts of the nervous system. Though these have been frequently differentiated into several distinct types, English writers have generally applied the term to oscillations of the eyes without distinction in regard to the variety of the movements. In this analysis no patient has been classified in the group without nystagmus if jerks were discovered at any time exceeding physiologic limits.

CASES OF CEREBELLAR TUMORS WITHOUT NYSTAGMUS

Of fifty-one cases of verified subtentorial new growths, there were eleven in which no nystagmus was discernible previous to operation. Nystagmus was absent in one additional case on repeated examination up to the day of exploration (table, 357). For purposes of comparison these are presented in the accompanying table.

The lesion concerned in eight of the cases was a glioma or a gliomatous cyst. In three there were tuberculomas, two being solitary. As regards the site of the new growths, it is seen from the table that they involved practically all portions of the cerebellum. Either the right or left hemisphere was affected as a whole or in part in six cases. The tenth and eleventh patients showed an involvement of both the vermis and an entire hemisphere. In the twelfth the lesion affected the deeper, anterior portion of the vermis and the right lobe. It is noteworthy that in no instance was the cerebellopontine angle primarily involved.

The degree of intracranial pressure varied widely among these patients. From a case with no measurable swelling of the optic nerve head and only moderate tension at operation, the pressure ranged to one with

4. Wilson and Pike: The Mechanism of Labyrinthine Nystagmus and Its Modification by Lesions in the Cerebellum and Cerebrum, Arch. Int. Med., January, 1915, p. 31.

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advanced choked disk and extreme herniation of the cerebellum into the spinal canal.

Accessory measures for eliciting nystagmus were tried in three of the patients—moving the head as first suggested by Oppenheim,[5] and using opaque spectacles as recommended by Bárány.[6] No success attended any of these tests.

The importance of making repeated observations is well shown by Case 357, in which a fine nystagmus finally appeared just previous to the operation.

The records of two patients illustrate how nystagmus which has been absent during one period of study may appear in later examinations subsequent to a suboccipital operation. Patient 30,000 on the second admission and Patient 357 on the second and third admissions had characteristic rhythmic movements of the eyes.

Two of the patients complained of tinnitus and three showed a slight homolateral reduction of air conduction. These complications had appeared during the present illness in each case. Functional tests, however, disclosed no serious impairment of the eighth nerve in any instance.

CASES OF CEREBELLAR AND EXTRACEREBELLAR TUMORS WITH NYSTAGMUS

While tumors may arise in every possible position in the posterior fossa, it is convenient for purposes of localization and surgical treatment to consider them as occupying four principal regions, two within and two external to the cerebellum. In regard to the former we may speak, first, of the tumors which occupy a central position, that is, those which involve the vermis and more or less of the median portion of each lateral lobe; and, second, of those which lie wholly within one or the other hemisphere. In reference to the latter we may look on the new growths either as occupying

5. Oppenheim: The Influence of the Position of the Head on Cerebral Symptoms, Neurol. Centralbl., 1910, xxix, 114.

6. Bárány: Functional Examination of the Vestibular Apparatus, Verhandl. deutsch. otol. Gesellsch., 1911, xx, 37.

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the cerebellopontine angle, or as lying more or less between the posterior portions of the lateral lobes, dorsalward from the medulla and vermis.

There were thirty-nine verified cases of tumors occupying one or the other of these regions in which nystagmus was present previous to operative treatment. In twenty-two the new growths were intracerebellar; ten occupied a median position, while eleven lay in one lateral lobe. In one there were gliomatous cysts in both hemispheres. The size of the new growths varied greatly, from lesions with a diameter of a few centimeters to growths which replaced most of the cerebellum. An extracerebellar tumor was found in each of seventeen patients. Of these tumors thirteen occupied a unilateral cerebellopontine site and three a posterior, median position. In one additional case an infiltrating glioma of the vermis lay partially exposed in the posterior, median sulcus.

The degree of intracranial pressure in this group was found to vary within wide limits. In several instances the eye-grounds appeared to be practically normal. Operation disclosed only a moderate tension of the suboccipital structures. In the balance of the cases, on the other hand, the changes in the optic nerve-head varied from a few diopters swelling to an advanced choking of the disk. A number of the patients showed marked secondary atrophy. At operation corresponding differences were noted in the degree of internal hydrocephalus.

The experiences with the caloric test and the pointing reaction (Bárány) in these patients have been discussed in another place.[7]

THE PRESENCE AND ABSENCE OF NYSTAGMUS IN CASES OF TUMORS LYING ANTERIOR TO THE CEREBELLUM

Nystagmus is sometimes found in patients with new growths lying anterior to the cerebellum. As its presence here is always confusing, this has somewhat

7. Grey: Studies on the Localization of Cerebellar Tumors: II. The Pointing Reaction and the Caloric Test, Am. Jour. Med. Sc., 1915, to be published.

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Patient	Lesion	Intracranial Pressure	Tinnitus and Hearing	
2320 ♂ 22	Tuberculoma involving vermis principally; rt. hemisphere slightly. (Operation.) Tumor about 3 cm. in diameter.	Choked disks 4-5 D. Marked tension at operation. Extreme herniation of cerebellum into spinal canal. Int. hydrocephalus.	No tinnitus. Hearing good. Slightly less acute rt. ear.	Some obj
1406 ♂ 45	Tuberculoma of left hemisphere. (Necropsy.) Tumor about 3 by 2.5 by 2 cm.	Choked disks 6-7 D. Marked tension at operation. Herniation into spinal canal.	No tinnitus. Hearing less acute in left ear.	None
320 ♀ 6½	Glioma involving vermis and medial portion of each hemisphere. (Operation and necropsy.) Tumor 4.5 by 3.2 cm.	Advanced choked disks. Marked cerebellar herniation into spinal canal. Int. hydrocephalus. Pons and medulla distorted.	No tinnitus. Hearing normal.	None
27788 ♂ 41	Cyst involving left hemisphere principally. (Operation.) Cavity about 5 by 4 by 4 cm.	Choked disks. Moderate tension of dura and cerebellum at operation.	No tinnitus. Hearing normal.	(?) ..
1444 ♂ 43	Multiple small tuberculomas involving vermis and left hemisphere. (Necropsy.) Largest 1 cm. in diameter.	Early choked disks 1 D. Moderate tension at operation. Probably slight int. hydrocephalus.	No tinnitus. Hearing about normal.	Marke rota self.
1642 ♀ 64	Cyst (gliomatous ?) involving rt. hemisphere principally. (Operation.)	Very early changes in eye-grounds. Hyperemic. No elevation. Moderate tension at operation.	No tinnitus. Hearing normal.	Marke obje
51 ♂ 8	Cyst (gliomatous ?) involving rt. hemisphere principally. (Operation.) 42 c.c. fluid from cyst.	Choked disks with secondary atrophy. Int. hydrocephalus marked. Separation sutures. Marked tension at operation. Great herniation into spinal canal.	No tinnitus. Hearing normal.	None
107 ♀ 3½	Cyst (gliomatous) involving rt. hemisphere principally, also vermis. Includes nearly whole rt. hemisphere. (Operation.)	Early choked disks. Marked int. hydrocephalus. Great herniation into spinal canal.	Tinnitus (?). Hearing about normal.	(?) ..
30000 ♀ 6	Large cyst (gliomatous) occupying left hemisphere principally. (Operation.)	Choked disks 5-6 D. Int. hydrocephalus. Marked herniation into spinal canal.	No tinnitus. Hearing less acute in rt. ear.	None
357 ♂ 36	Glioma involving anterior portions, rt. hemisphere and vermis. (Operation.)	Choked disks 5 D. Marked tension at operation. Herniation into spinal canal.	Some tinnitus each ear. Hearing normal.	Fairly tatio self.
151 ♀ 9	Large cystic glioma involving rt. hemisphere principally, also vermis. (Operation.)	Advanced secondary optic atrophy. No elevation. Hydrocephalic configuration of skull. Marked pressure at operation. Int. hydrocephalus.	No tinnitus. Hearing normal.	None
26387 ♀ 37	Cystic glioma involving left hemisphere principally. (Operation.)	Choked disk. Moderate tension at operation. Int. hydrocephalus.	Considerable tinnitus rt. ear. Later left ear. Hearing normal.	Some

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RS AND NO NYSTAGMUS

ness	Eye Muscles	Caloric Test	Nystagmus
rotation of self.	No strabismus. Conjugate deviations good. Very slight restriction toward pt's. left.	Definite nystagmus from irrigation either ear. More marked from left. Normal pointing reactions. Reaction movements from both sides.	None with eyes directed forward, to either side, up and down, on changing position of and shaking head, or on breaking fixation.
.....	No strabismus. Conjugate deviations good.		None with eyes directed forward, to either side, or up and down.
.....	No strabismus. Conjugate deviations good.		None with eyes directed forward, to either side, or up and down.
.....	Slight weakness left internal rectus.	Definite nystagmus from irrigation either ear. Normal vestibular response, both sides.	None with eyes directed forward, up and down, or to the right. A few atypical jerks on looking to the left.
iness. No objects or	No diplopia or strabismus. Some weakness left ext. rectus (?).	Marked nystagmus within very short time from irrigation either ear. Normal pointing reactions and reaction movements.	In two examinations atypical jerks of brief duration appeared only when looking to either side. Subsequently no jerks or nystagmus on directing eyes ahead, to the sides, and up and down. None on moving head and on breaking fixation.
rotation of self.	No diplopia or strabismus. Conjugate deviations good.	Marked nystagmus within very short time from irrigation either ear. Normal pointing reactions and reaction movements.	None with eyes directed forward, to either side, up and down, on moving head, or on breaking fixation.
.....	Some diplopia. No strabismus. Conjugate deviations good.		None with eyes directed forward, to either side, or up and down.
.....	No diplopia or strabismus. Conjugate deviations good.		None with eyes directed forward, to either side, or up and down.
.....	Paralysis left abducens. Very slight weakness rt. abducens (?).		None with eyes directed forward, to either side, or up and down.
d. No ro- objects or	History of diplopia. No strabismus. Conjugate deviations good.	Slight nystag. from rt. ear; marked from left. Reaction movements only from left ear. Pointing errors to right both arms.	None with eyes turned in all directions on numerous examinations. Just previous to operation a fine, indefinite nystagmus appeared on looking to either side.
.....	No diplopia. No strabismus. Conjugate deviations good.		None with eyes directed forward, upward, or to her right. Some atypical coarse jerks on looking toward her left.
.....	No diplopia. No strabismus. Conjugate deviations good.	No nystagmus provoked on irrigating either ear. No subjective distress.	None with eyes turned in all directions.

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limited its usefulness as a sign of disease in the posterior fossa. The types of nystagmus in the two classes (that is, patients with tumors lying anterior to the cerebellum and those with cerebellar or extracerebellar growths) are occasionally identical. In each the movements may be fine and inconspicuous or they may appear as coarse, well-marked jerks. It is frequently impossible, even, to recognize the variety arising from direct irritation in the labyrinth; for, as Friesner and Braun[8] have pointed out, despite much that has been said to the contrary, the nystagmus due to cerebellar disease often cannot be differentiated clinically from that due to labyrinthine disease. Our experience has been that on the average the movements are coarser and more marked in individuals with lesions situated below the tentorium.

The fact that no nystagmus was noted in fully one third of the cases of intracerebellar new growths suggested a similar analysis of the cases with verified lesions lying anterior to the cerebellum in order to determine the relative frequency of this sign in the latter group.

Of forty patients with tumors situated anterior to the cerebellum, eight showed characteristic rhythmic oscillations of the eyes previous to any surgical interference. The new growths and their locations were as follows: one glioma, right occipital lobe; one glioma, right thalamus principally; one gumma, left temporal lobe; two cases of gliomatous cyst, one right and one left, parietal lobes; one solitary tubercle and one glioma, pons; and one hypophysial cyst.

The caloric test was used on three of the patients—occipital lobe glioma, thalamic glioma, and pontine tubercle. In each a typical nystagmus appeared following the irrigation of either ear.

There were thirty-two individuals in whom no nystagmus was detected; one gumma and one glioma,

8. Friesner and Braun: The Functional Tests of the Static Labyrinth in Neurological Diagnosis, New York Med. Jour., 1914, c, 369.

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left frontal lobe; one gliomatous cyst, right frontal lobe; one endothelioma, pons; one glioma, left thalamus; two cases with a glioma each, and two with an endothelioma each, right parietal lobe; one glioma, left parietal lobe; two patients with a glioma each, and one with a gumma, left temporal lobe; one gliomatous cyst, one glioma, and one endothelioma, right temporal lobe; one glioma involving both thalami, right principally; one endothelioma, right temporoparietal region; one endothelioma at the foramen magnum; one metastatic melanotic sarcoma—multiple in cerebrum and cerebellum; and eleven patients with hypophysial strumas or cysts.

Seven of the patients were examined with the caloric test. Typical nystagmus appeared following the irrigation of each ear in six of these: unilateral thalamic glioma, frontal gumma, bilateral thalamic glioma, temporal endothelioma, endothelioma at foramen magnum, and parietal endothelioma. In the seventh—a pontine endothelioma—there was no response from either labyrinth. An involvement of the third, fourth and sixth nerve nuclei probably was largely responsible for this, as voluntary conjugate deviation of the eyes was practically impossible in all directions.

A study of the eye-grounds showed no uniformity in the ophthalmoscopic pictures. The changes in the nerve-head varied in both classes of cases (patients with and patients without nystagmus) from fundi with no measurable elevation to advanced choked disk. A similar variation was pointed out in discussing the cases of intracerebellar new growths (table, column 4).

DISCUSSION

In seeking an explanation for the presence or absence of nystagmus in patients of this class it is necessary to consider briefly the present conceptions regarding the mechanism of nystagmus. These, unfor-

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tunately are somewhat discordant. Bauer and Leidler[9] believe from their experiments on animals that nystagmus appears as a result of lesions in the region of Deiters' nucleus—the horizontal jerks more from the ventrocaudal part, and the vertical jerks from the oral part. In a similar way Bárány[10] assumes a nystagmus center here and explains the presence of this and certain other so-called cerebellar signs as the result of increased tension in the posterior fossa. Tumors in any location, as soon as they exert pressure on the posterior fossa, may give rise to an intracranial type of nystagmus, which may be transitory and of varying intensity or permanent and of constant intensity. Hesse,[11] however, questions whether we are justified at present in attributing such signs to the distant effects (*Fernwirkung*) of space-consuming new growths in the large and small brain.

Wilson and Pike,[4] on the other hand, do not subscribe to this or to the various other hypotheses which recognize a nystagmus center. They conclude that an agency somewhere produces a deviation of the eyes from the primary position of equilibrium. In labyrinthine nystagmus this agency lies in the labyrinth. The internal rectus muscle on the side of the slow deviation and the external rectus on the side opposite are subjected to a greater strain than usual. This strain, resulting in the stimulation of the sensory or afferent endings of the eye muscles, sets up afferent impulses which are conveyed to the cerebrum over the afferent fibers in the third, fourth, and sixth cranial nerves.[12] These afferent impulses, in their turn, set up efferent impulses in the oculomotor cells of the

9. Bauer and Leidler: The Effect on the Vestibular Eye Reflex of Excising Different Portions of the Brain, Arb. a. d. neurol. Inst. a. d. Wiener Univ., 1912, xix, 155.

10. Bárány: Further Investigations on the Relations of the Vestibular Apparatus to the Central Nervous System, Wien. med. Wchnschr., 1912, lxii, 3210.

11. Hesse: The Rotation and Caloric Nystagmus, Ztschr. f. Neurol. u. Psych., 1913, xv, 377.

12. Tozer and Sherrington: Receptors and Afferents of the Third, Fourth and Sixth Cranial Nerves, Folia Neuro-Biol., 1910, iv, 626.

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cerebrum, which result in a quick, jerky contraction of the internal rectus on the side of the slow deviation and of the external rectus of the opposite side, with relaxation of the antagonistic muscles. The effect is a restoration of the eyes to the primary position. The complex motor mechanism of the eyes, these authors conclude, cannot be described in terms of circumscribed isolated centers, but must be defined in terms of an integrative system.

Mills and Weisenburg[13] regard the nystagmus seen with cerebellar lesions as due to an involvement of the vestibulocerebellar apparatus, probably of the parts connecting the vestibular nuclei with the deep nuclei of the cerebellum.

We are acquainted with the agency connected with the labyrinthine type of nystagmus of which Wilson and Pike speak, but as yet are more or less unfamiliar with that responsible for the development of nystagmus from other portions of the brain. Just what relation the cerebellum bears to the development of rhythmic movements of the eyes is at present rather uncertain. The cerebellum, it appears, has no pathways or centers indispensable for the appearance of either the slow or the quick component. From the investigations of Bauer and Leidler[9] it is clear that while destruction of parts of the cerebellar hemisphere and vermis never causes spontaneous nystagmus, lesions of the latter, nevertheless, result in an excessive excitability of the vestibular nuclei. This has led these workers, with Bárány, Neuman and others, to the belief that in cerebellar disease the nystagmus is due to an alteration in function of the vestibular nuclei or their connections with the nuclei of the eye muscles through pressure.

Oppenheim,[14] with Bruce, Wallenberg, Adler, Probst, Babinski, Horsley and others, assumes a

13. Mills and Weisenburg: Cerebellar Symptoms and Cerebellar Localization, THE JOURNAL A. M. A., Nov. 21, 1914, p. 1813.
14. Oppenheim: Lehrbuch der Nervenkrankheiten, 1913, p. 1383.

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nystagmus of cerebellar origin. Horsley,[15] for example, has shown that a careful electrical stimulation of the white matter in the neighborhood of the dentate nucleus, or of the body of the dentate nucleus itself, provokes a horizontal nystagmus toward the side excited. According to this view, as Babinski[16] has pointed out, nystagmus can be caused by either a labyrinthine or a cerebellar irritation. Rothmann,[17] on the other hand, from excitation experiments of his own, is led to dispute such an influence of the cerebellum on the eye muscles.

In considering the various hypotheses which have been advanced to account for the appearance of nystagmus in patients with intracranial tumors, it is evident that there is nothing in these views which will satisfactorily explain the absence of nystagmus in the series reported here. It has already been pointed out that of thirty-four patients with verified intracerebellar new growths, at least one third showed no nystagmus. Now in both of these groups—that without rhythmic movements of the eyeballs as well as that with them—the same differences were found in the degree of intracranial tension. In both there were patients with very little changes in the eye-grounds and with only moderate tension of the cerebral structures at operation; there were also patients with advanced choked disks and marked herniation of the cerebellum into the spinal canal. A similar variation was noted in the situation of the new growths.

On consulting the table it is clear that the absence of this sign cannot be accounted for by a paralysis of the eye muscles. In eight of the twelve patients practically normal conjugate movements were possible in all directions. Three showed a slight weakness of one external rectus muscle, but this degree of paresis is

15. Horsley, quoted by Babinski and Tournay (Note 16).
16. Babinski and Tournay: The Symptoms of Diseases of the Cerebellum and Their Significance, Tr. Internat. Med. Cong., London, 1913, Sec. XI, Part 1.
17. Rothmann, quoted by Oppenheim (Note 14).

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very frequently noted in patients who show characteristic oscillations of the eyeballs. In only one instance was a complete paralysis of the abducens found. Parallel movements of the eyes were possible here in each of the other directions.

Probably the most important evidence that the absence of nystagmus was not due—in a number of these cases at least—to an impairment of the fundamental mechanism of nystagmus is afforded by the results from the caloric tests. Caloric examinations made in six of the twelve patients showed well-marked nystagmus following the irrigation of each labyrinth in five.

On contrasting the group of subtentorial tumor cases having nystagmus with the group showing no nystagmus, it is evident that rhythmic oscillations of the eyes were found in every patient with an extracerebellar new growth. It should be mentioned, however, that the nystagmus was rather inconspicuous in the cases of posterior, median growths. In one instance the fine, rapid jerking of the eyeballs was noticed only when the patient looked toward his right side. The nystagmus in all of the cerebellopontine cases was well marked. Though the number of cases used in this study is comparatively small, one feature is sufficiently prominent to suggest a generalization. The results from the analysis would seem to indicate that when a patient exhibits a cerebellar tumor syndrome without nystagmus, the absence of rhythmic movements of the eyes points toward an intracerebellar localization of the lesion.

There are several explanations which assist in accounting for the presence and absence of nystagmus in patients with supratentorial new growths. Bárány[10] believes that in such cases it is due to pressure exerted on the nystagmus centers—in the region of Deiters' nuclei—by the tumors from a distance (*Fernwirkung*). When the growths are not space-consuming, however,

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and lead to no increased intracranial tension, rhythmic oscillations of the eyes do not appear. Another suggestion is offered by Bauer and Leidler.[9] They have shown experimentally that after cerebral hemisphere extirpations there is an increased excitability of the corresponding eye muscle nuclei. It is conceivable that certain cerebral tumors may lead to such a result.

Certain experiments of Wilson and Pike[4] are interesting in this connection. They removed various areas of the cortex, frontal, parietal, occipital, and temporal and also destroyed the subcortical ganglionic masses in cats, dogs and monkeys in an attempt to find some region in a hemisphere where a lesion will modify or abolish labyrinthine nystagmus. The only place where they obtained consistent alteration of the labyrinthine stimulus, and that only so far as the quick component was concerned, was in the region of, or adjacent to, the temporal lobe.

A review of the clinical findings in the patients with supratentorial tumors shows that both the quick and the slow phases of nystagmus may exist in patients with new growths occupying very diverse regions in the cerebrum. In six of the seven patients examined with the caloric test (no spontaneous nystagmus) characteristic rhythmic movements of the eyes appeared following the irrigation of either meatus. In the seventh patient there was almost a complete ophthalmoplegia. Similar results were obtained from patients who showed spontaneous nystagmus. The caloric test was used in three of this group and in each case typical nystagmus followed the stimulation of either labyrinth.

SUMMARY

Of the thirty-four verified cases of cerebellar tumor and seventeen verified cases of extracerebellar tumor, eleven showed no nystagmus previous to operation on repeated examination. All of the cases in which it was absent contained intracerebellar new growths—

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32 per cent. of the intracerebellar series. This suggests that when a patient exhibits a cerebellar tumor syndrome without nystagmus, the absence of rhythmic movements of the eyes points toward an intracerebellar localization of the lesion.

Among the members of both groups (that is, the group with and the one without nystagmus) there were marked differences in the degree of intracranial tension. In each group there were new growths which involved the vermis and others which replaced a part or the whole of one or both hemispheres.

Caloric examinations made in six of the cases in which there were no rhythmic movements of the eyes resulted in characteristic nystagmus from either labyrinth in five.

Of forty verified cases of tumors lying anterior to the cerebellum, eight showed nystagmus previous to operation. Caloric examinations made in seven of the cases without nystagmus provoked characteristic rhythmic oscillations of the eyes from either labyrinth in six.

These results indicate that in many cases of intracranial tumor the absence of nystagmus cannot be accounted for by an impairment of the fundamental mechanism of nystagmus.

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GALVANOMETRIC STUDIES OF THE CEREBELLAR FUNCTION*

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The object of this study has been to ascertain, if possible, the mechanism of the cerebellar function, as deduced from the phenomena consequent to its lesions. It will be necessary, therefore to formulate at the outset a clear conception as to which of such phenomena are essentially cerebellar in origin. The cerebellum is adjacent to so many important structures and forms such intimate connections with the vestibular system and its oculomotor tracts that many of its lesions, experimental, as well as clinical, but especially the latter, very frequently implicate these other structures, producing their corresponding symptoms. On reviewing the literature we find, therefore, that a number of symptoms have from time to time been ascribed to this organ which have not had their genesis in it at all.

THE CEREBELLUM AND SENSORIUM

Lesions of the cerebellum do not produce disturbances of sensibility, and if these are present, they are invariably due to the involvement of the sensory tracts or nerves found in its vicinity. This is attested by such a great number of observations that it may well be considered an indisputable fact. There have been no sensory disturbances in the experimental lesions of this organ produced by Rolando, Flourens,[1] Dickin-

* From the Hull Physiological Laboratories, University of Chicago.
1. Flourens: Recherches expérimentales sur les propriétés et les fonctions du système nerveux, 1842, Ed. 2, p. 37.

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son,[2] Leven and Ollivier,[3] Luciani,[4] and Ferrier.[5] In cerebellar disease of man, sensory disturbances are denied by Akland and Ballance,[6] Holmes and Stewart,[7] André-Thomas[8] and others. Risien Russell[9] and Lewandowsky,[10] especially the former, differed from the above-named observers and maintained that unilateral ablation of the cerebellum is followed by anesthesia and analgesia on the side of the lesion. The great difficulty of determining sensory loss in animals detracts, however, a great deal from the value of their observations, and the weight of evidence on this point rests rather with the clinician than with the physiologist.

What was said with regard to general sensibility applies with equal force to that type of sensation which is implied by the general term of the "muscular sense." As is well known, this type of sensation comprises all those sensory reactions which, originating in the motor organs and their accessories (tendons, joints, etc.), provide us with information as to their state of tension, their direction in active and passive movements, and incidentally aid us in the appreciation of our posture in space. The cerebellum has been thought to be the center of the muscle sense by such noted observers as Lussana,[11] Hitzig, Munk[12] and of late Lewandowsky, and the spinocerebellar tracts have been identified by them as the carriers of the so-called proprioceptive stimuli. It is an undeniable fact, how-

2. Dickinson, W. H.: Brit. and Foreign Med. Chirurg. Rev., 1865, xxxvi, p. 455; Boston Med. and Surg. Jour., 1865, lxxi, 239 (abst.).
3. Leven, M., and Ollivier, A.: Arch. gén. de méd., 1862, ii, 519; ibid., 1863, i, 78.
4. Luciani, L.: Il Cerveletto, Florence, 1891; Das Kleinhirn, Ergebn. d. Physiol. (Asher-Spiro's), Wiesbaden, 1904, iii, Abt. II.
5. Ferrier, D.: Functions of the Brain, Ed. 2, 1886, p. 174, etc.
6. Akland, T. D., and Ballance, C. A.: St. Thomas Hosp. Rep., London, 1896, xxiii, 163.
7. Holmes and Stewart: Brain, 1904, xxvii, 522.
8. André-Thomas: Cerebellar Functions, Engl. Transl. by W. C. Herring, New York, 1912.
9. Russell, J. S. Risien: Phil. Tr. Roy. Soc., London, 1895, clxxxv B, 819.
10. Lewandowsky, M.: Die Funktionen des zentralen Nervensystems, Jena, Verlag v. G. Fischer, 1907.
11. Lussana, F.: Jour. de la physiologie de l'Homme, 1862, v, 418; vi, 1863, 169, etc.
12. Munk, Quoted by André-Thomas: Cerebellar Functions, English Trans. by W. C. Herring, New York, 1912.

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ever, that pure cerebellar lesions do not give rise to loss or impairment of the muscle sense and, if this is present, there is invariably found an associated lesion of the posterior columns or their nuclei.[18] Patients with cerebellar disease have complete orientation toward the surrounding world, are fully conscious of all changes in posture during active and passive movements and present no Romberg sign (swaying on being deprived of the aid of vision). As pointed out by Luciani, the dog deprived of a lateral lobe of the cerebellum even anticipates the disturbances that will ensue on walking or prehension of food, and to obviate a fall seeks out some support for the side of the lesion.

THE CEREBELLUM AND MOTILITY

The phenomena consequent to cerebellar lesions belongs to the motor sphere and not to the sensory. This is almost universally conceded now by both physiologists and clinicians. The disturbances manifest themselves, however, in such a large variety of phenomena, owing chiefly to concomitant lesions of other structures, that their interpretation has for a long time been exceedingly difficult. Of late, however, thanks to our increasing knowledge of the anatomy and physiology of the cerebellum as well as of its adjacent structures, this difficulty has been largely removed, and we are able to dissociate the true cerebellar phenomena from the others.

FORCED MOVEMENTS, NYSTAGMUS, ETC.

It is pretty definitely established that such phenomena as forced movements, the circus movements and rolling movements in animals and the so-called imperative movements in man, are not cerebellar in origin. And the same is true of the nystagmus, the conjugate deviation of the eyes and the characteristic attitude of the head so frequently observed after

13. André-Thomas: Cerebellar Functions, p. 144.

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unilateral ablation of the cerebellum in animals and occasionally in cerebellar disease of man.

These phenomena are essentially vestibular in origin, being due to a lesion of the vestibular complex itself or its oculomotor tracts. Muskens,[14] who made an extensive study of these phenomena in their relation to the pathologic findings at necropsy, found that they are associated with lesions in the paracerebellar nuclei, the vestibulomesencephalic tracts in the posterior longitudinal bundle and the descending vestibulospinal tracts. They are, at any rate, not cerebellar symptoms. The whole lateral lobe of the cerebellum may be removed without producing the slightest indication of these phenomena. I have personally performed such operations in about twenty-five animals (dogs and cats) and can say without hesitation that forced movements as well as nystagmus are not cerebellar in origin. I have seen no instance of rolling movements or a tendency to it in pure cerebellar lesions. They were, however, extremely vehement and almost uncontrollable whenever there was an associated lesion of the vestibular system. These movements in my cases were invariably toward the side of the lesion in accordance with the results of Schiff,[15] Ferrier and Turner,[16] André-Thomas, and Muskens[14] in lesions of the cerebellum, and those of Bechterew[17] and Breuer[18] after section of the eighth nerve.

THE PHENOMENA OF CEREBELLAR LESIONS

The phenomena that are purely cerebellar in genesis are two, the ataxia and the tremor. I am using the term "ataxia" in its general sense to denote irregularity or perversion of muscular action without reference to its nature, and in contradistinction to loss of movement such as is found in paralysis. Personally I have never

14. Muskens, L. J. J.: Jour. Physiol., London, 1904, xxxi, 212; Brain, 1914, xxxvi, 352.
15. Schiff, M.: Lehrbuch der Physiologie, 1858, I, 354.
16. Ferrier and Turner: Phil. Tr. Roy. Soc., London, 1894, clxxxv, B, p. 730.
17. Bechterew: Arch. f. d. ges. Physiol., 1882, xxx, 318.
18. Breuer: Arch. f. d. ges. Physiol., 1891, xlviii, 250.

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been convinced as to the existence of hypotonia in cerebellar lesions, a symptom on which Luciani laid so much stress. In my experience, which is in accord with that of Ferrier, it is very inconstant. I have noted it only in very extensive lesions, in lesions which have involved also the vermis and its nuclei. The nucleus tecti, as established by the morphologic studies of Weidenreich[19] and others, may be looked on as a part of the paracerebellar nuclei, which, from their rôle in the production of decerebrate rigidity (Thiele[20]), are known to dominate the tonicity of the body musculature. The presence of hypotonia in such severe lesions of the cerebellum is thus easily explained without attributing it to a disturbance of the chief function of the organ. The ataxia and tremor, on the other hand are constant and marked, and found in lesions limited to the lateral lobes and in the absence of any involvements of the vestibular complex. On reviewing the literature of the many cases of agenesis of the cerebellum, such as have been reported by Edinger and Neuberger,[21] Andral,[22] Hitzig and others, it is noted that while the patients have shown practically no evidence of disease during life, having had normal strength, normal sensibility and no disturbances of equilibration, they have invariably shown some degrees of ataxia and tremor. And it is to interpret these phenomena that our efforts should be directed in attempting to decipher the complex and mysterious problem of the mechanism of the cerebellar function.

THE INTERPRETATION OF THESE PHENOMENA

There have been numerous theories advanced in explanation of these phenomena. Flourens, Vulpian,[23] Renzi and Dickinson interpreted them as disturbances of coordination, attributing to the cerebellum that function which, according to Foerster's definition of

19. Weidenreich: Zeitschr. f. Morphol. u. Anthropol., 1899, i, 259.
20. Thiele, F. D.: Jour. Physiol., London, 1905, xxxii, 355.
21. Neuberger and Edinger: Berl. klin. Wchnbl., 1898, No. 4, p. 69.
22. Andral: Clinique médicale, Ed. 4, v, 713.
23. Vulpian: Leçons sur la physiologie générale et comparée du Système Nerveux, Paris, 1866.

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coordination, enables the organism by means of its muscles to produce external effects which fulfil a definite purpose or a set task, accomplishing these effects with the smallest possible waste of energy. Bouillaud,[24] Ferrier and Thomas thought the cerebellum to be an organ for the maintenance of body equilibrium and endeavored to explain the phenomena of the lesions on that hypothesis. Babinski,[25] basing his opinion on clinical studies, proposed the theory of asynergia of movements, according to which it is essentially lost in cerebellar lesions in the harmonious association of simple movements necessary for the execution of a complicated movement and he endeavored to prove it by certain clinical tests. Babinski admitted, however, that in addition to asynergia there is also a dysmetria which is the cause of the excessive and ill-proportioned movements in cerebellar lesions. As to the asynergy, it is of course clear that it is merely a broad, general term for incoordination, and this conception of the nature of the disturbances aids us but little in understanding the mechanism of their production.

According to Holmes and Stewart the cerebellar phenomena are the result of defective cooperation of muscles and their antagonists. The cerebellum, according to this view, effects relaxation of the extensors while the flexors are in a state of contraction, and relaxation of the flexors while extension is in progress, the same being applied to the abductors and adductors.

OBJECTIONS TO THE FOREGOING VIEWS

There are two points in common to all these theories: First, they all ascribe to the cerebellum a function which is totally distinct from that possessed by any other part of the nervous system, a function that is closely allied with motor activity but still different from it in a vague, indefinite way. Secondly, the function of the cerebellum, according to any of these

24. Bouillaud: Arch. gén. de méd. 1827, xv, 64.
25. Babinski, J.: Rev. neurol., Nov. 9, 1899, p. 784; Feb. 2, 1901, p. 260; April 18, p. 422; May 15, 1902, p. 470; Nov. 15, 1902,

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theories, cannot be expressed in the terms of a single neuron effect. It is of course evident that coordination of movement, synergy of movement, etc., an effect which manifests itself simultaneously in a large group of muscles, widely isolated from each other and differing in their mode of action, cannot be the expression of an individual neuron. It must be the resultant effect of a large number of neurons all functioning at the same time. This conception of function is, however, entirely unnecessary for our understanding of the activity of any other nerve structure, in which case each unit, whether motor or sensory, may exhibit its individual function independently of the other neurons and produce a corresponding effect.

THE THEORY OF LUCIANI

The theory which is perhaps best known and most widely discussed, and to which the foregoing objections do not apply, is that of the famous physiologist Luciani. The cerebellum, according to him, is a small system which is relatively independent of the cerebrum and cord, and its function is that of a great coadjutor and reinforcer of the whole cerebrospinal nervous system. The cerebellum, Luciani maintained, does not exhibit a function peculiar to itself; its function is exclusively that of augmenting the other centers, lending them strength (sthenic action), tonicity (tonic action) and effecting proper fusion of the cerebral stimuli (rhythmic action). Loss of cerebellar innervation results, therefore, as soon as the early irritative phenomena have passed off, in asthenia or weakness, atonia or relaxation and flabbiness of the muscles and arrhythmia with the resultant tremor. The phenomena of cerebellar lesions are, according to this theory, purely motor in character, analogous, although not entirely identical with, those following destruction of the rolandic zone of the cortex or of the anterior horn cells in the spinal cord.

While loss of cerebellar innervation results, according to Luciani, only in diminution of strength, some

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authors went even further and postulated a true cerebellar paralysis. Notable among these was Risien Russell, who maintained that unilateral ablation of the cerebellum was in his experiments followed by genuine paralysis on the side of the lesion, entirely identical with that following ablation of the motor area of the cortex. This is however denied by the majority of observers. There wa no paralysis in the animals operated on by Bouillaud,[24] Dalton,[26] Longet,[27] Munk and Ferrier and Turner.[15] Personally, I am convinced that pure cerebellar lesions do not give rise to paralysis. One has only to compare the gait of an animal after destruction of the motor area of the cerebrum with that which follows unilateral ablation of the cerebellum to be impressed by the marked dissimilarity between these two conditions. In the former instance, the limbs on the side opposite to the lesion are distinctly paretic; they are unable to support the animal on that side; they bend under it and are frequently placed with the dorsal surface to the ground. In the latter condition, on the other hand, the limbs on the side of the lesion are generally in full extension; they are never placed with the dorsal surface to the ground, but are usually thrown violently forward or outward, and the frequent falls are therefore to be attributed to the ill-proportioned activity of the muscles rather than to loss of their activity.

Clinically, paralysis in cerebellar disease is likewise denied by Macewen,[28] Oppenheim,[29] André-Thomas[8] and others. That there is no true paralysis in cerebellar lesions is also indicated by the state of reflexes. They are not at all characteristic. They are sometimes increased and sometimes diminished and very frequently perfectly normal. The increase or diminution, when present, is not limited to the side of the lesion,

26. Dalton, J. C.: Am. Jour. Med. Sc., 1861, p. 84.
27. Longet: Traité de physiologie, Ed. 3, iii, 466.
28. Macewen, W.: Infective Diseases of the Brain and Spinal Cord, Glasgow, 1893, p. 195.
29. Oppenheim, D.: Diseases of the Nervous System, English Transl. by C. C. Mayer, Ed. 2, p. 699.

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but is found, and in the same degree, on the healthy side as well.

This has been determined experimentally by Ferrier and Turner[15] and clinically in the forty cases of cerebellar tremor described by Holmes and Stewart.[7]

The view that the cerebellum exerts a motor effect, which, while different in nature from that of the motor cortex, is in the same manner as the latter exerted directly on the periphery, has also been held by Luys[30] and developed by Hughlings Jackson.[31] The latter, basing his opinion on certain cases of cerebellar tremor with tetanus-like seizures observed by him, said:

> The cerebellum is the center for continuous movements, and the cerebrum for changing movements. Borrowing medical terms to describe normal physiological conditions, the former are tonic and the latter clonic.

He amplified this opinion later by saying that the cerebellum represents movements of the skeletal muscles in the order, trunk, leg and arm preponderatingly extensorwise; that the cerebrum represents movements of the arm, leg and trunk preponderatingly flexorwise, and that in continuously acting on the motor centers of the spinal cord the impulses from the cerebrum and cerebellum antagonize or inhibit one another in the same degree as the order of the degree of their different representations of movement of the muscles of the body. This view has also been championed by Horsley[32] and others.

GOWERS' HYPOTHESIS

Gowers,[33] as far as I can ascertain, was the only one to maintain, on purely theoretical grounds, that the cerebellum has no downward effect whatever, that it exhibits its influence on the cerebrum exclusively, this influence being inhibitory in character, and that cerebellar coordination is nothing else than coordination of

30. Luys: Recherches sur le système nerveux, Paris, 1865, p. 429.
31. Jackson, Hughlings: Med. Examiner, April 5, 1877; Med. Times and Gaz., 1878, ii, 485; Brain, 1906, xxix, 425.
32. Horsley, V.: Brain, 1906, xxix, 447.
33. Gowers, W. R., Lancet, London, 1890, i, 955.

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the activity of the rolandic cortex. His arguments in support of this theory were as follows: The cerebellum is connected with the periphery by means of afferent or upbearing tracts exclusively. The only efferent or outgoing tracts known to come from it pass by way of the superior cerebellar peduncle to the opposite hemisphere of the cerebrum, and it is inconceivable that an organ having no outgoing tracts to the periphery could exert a direct motor effect on it. Continuing he said:

> The will may alter any muscular contractions by which the upright posture is maintained, and yet without thus being deranged for an instance, and it is difficult to understand how this is possible if the two motor effects, the cerebral and cerebellar, take place apart, and equally difficult if we assume that they only come together in the subordinate centers in the spinal cord.

He therefore concluded that the cerebellum exhibits its function on the motor cortex of the opposite hemisphere of the cerebrum by way of its efferent tracts passing along the superior cerebellar peduncles, and that this function is inhibitory in character, in view of the fact that evidence at hand tends to show that when one set of cells exerts an influence on another set, this is in the nature of control, as witness the influence of the cortical motor cells on the spinal cells in the anterior horns. We must distinguish, said Gowers, between the continuous action of nerve cells, which by its nature needs control and restraint, and occasional action, which requires stimulation, and conceived as excitation.

According to this conception, the phenomena consequent on cerebellar lesions are only indirectly cerebellar; they are primarily cerebral effects, and, like the increased reflexes and spasticity after lesions of the upper motor nervous system, are due to removal of inhibition on the nerve cells directly concerned in their production.

It is evident from the foregoing that, according to Luciani, Hughlings Jackson and others, the distur-

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bances of motility in cerebellar lesions are the expression of diminished or lost functional activity on the side of the lesion; according to Gowers, on the other hand, they are the result of exaltation of such activity.

It occurred to me that the problem could be taken out from the realm of pure speculation and solved by means of the galvanometer.

SOLVING THE PROBLEM GALVANOMETRICALLY

It is well known that living tissue in a state of activity exhibits four kinds of phenomena, mechanical, chemical, thermal and electric. Of all these the electric phenomena are the most faithful; they are to be observed, especially so in the case of nerve tissue, when none of the others can be elicited.[34] Among the electric phenomena is the state of negativity which excited tissue assumes with relation to the quiescent part thereof. On connecting two such parts with a galvanometer, with the circuit closed, a current is seen to pass through it, as evidenced by the deflection of the needle, from the quiescent to the active part. The former is therefore in its external or galvanometric relations positive to the latter. In completing the circuit and passing through the tissue the current of course passes in the opposite direction, from the active to the resting part. Active tissue, in other words, behaves electrically toward quiescent tissue as the zinc element of a voltaic couple behaves toward the copper element — it is with regard to the outer or galvanometric circuit, negative to the latter. While this electric phenomenon, the so-called action current, is generally studied by means of the negative variation, or the diminution of the preexisting current of injury which accompanies the excitation of the tissue, the latter, says Hermann,[35] is only a special case of a more general phenomenon, which is, that active tissue is,

34. Waller, A. D.: Eight Lectures on the Signs of Life, New York, 1903, E. P. Dutton & Co., p. 10; On Skin Currents. Observations on Cats, Proc. Roy. Soc., 1901, lxix, 171

35. Hermann, L.: Handbuch der Physiologie, ii, part 1, p. 194; Borrutau, D.: Arch. f. d. ges. Physiol., 1901, lxxxiv, 95.

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properly speaking, invariably negative (or "zincative," according to Waller) to resting tissue quite independently of previous preexisting currents. The action current is also entirely independent of the agency which provoked the activity in the tissue. The stimulus may be chemical, mechanical, thermal or electric; it may be direct or indirect. Gotch and Horsley[36] have thus demonstrated that the action current manifests itself in a peripheral nerve, not only on direct excitation of the nerve, but also on stimulation of the contralateral and functionally related motor area of the cerebrum.

That natural nervous activity is, like that resulting from artificial stimulation, accompanied by the development of a negative state, has been demonstrated by A. Beck,[37] Alcock and Seemann,[38] Einthoven[39] and others. Beck removed the brain, spinal cord and sciatic nerves of a frog *en bloc* and placed the preparation on a glass plate. He then applied nonpolarizable electrodes to various parts of the nervous system and connected them with a galvanometer. He found that there was uniformly an electrical difference of such a nature that the proximal, more active parts of the nervous system were negative to the distal parts. Alcock and Seemann and Einthoven studied the impulses which traverse the vagus during natural respiration and found that a state of negativity invariably passes along the nerve with each inspiratory excursion. The latter also observed minor effects which accompanied the large waves of the inspiratory acts and corresponded with rhythmic contractions of the heart. A natural corollary to this should be that, if active tissue on one side of the body were connected by means of a galvanometer with the corresponding quiescent tissue

36. Gotch, F., and Horsley, V.: On the Mammalian Nervous System, Its Functions and Their Localization Determined by an Electrical Method, Phil. Tr. Roy. Soc. London, 1891, clxxxii, B, p. 332.
37. Beck, A.: Centralbl. f. Physiol., Nov. 8, 1890.
38. Alcock, N. D., and Seemann, J.: Arch. f. d. ges. Physiol. (Pflüger's), 1905, cviii.
39. Einthoven, W.: Ueber Vagusströme, Arch. f. d. ges. Physiol. (Pflüger's), 1908, cxxiv, 246.

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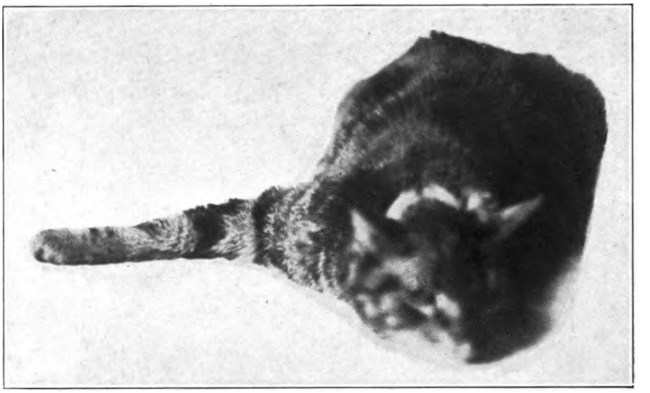

Fig. 1.—Cat after removal of left motor area of the cerebrum and right lateral lobe of the cerebellum. From photograph taken fifteen days after the first operation and fifteen days after the second. Note marked extension of right forelimb on prolonged standing. The extension is not due to a spasmodic condition of the forelimb, as it takes place gradually and is brought about by the weight of the body forcing the paretic limb out.

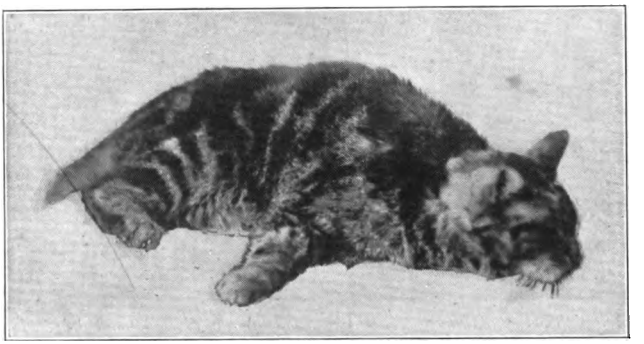

Fig. 2.—Same cat as shown in Figure 1. In standing posture for two or three minutes. Considerable abduction of both limbs on right, paretic side.

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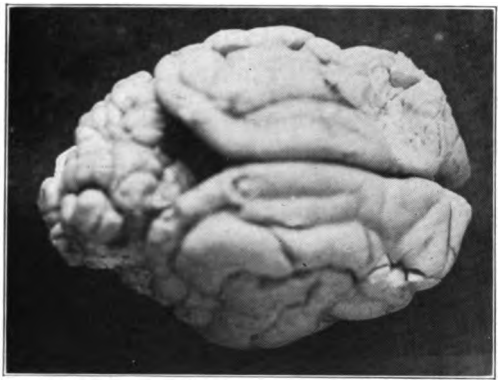

Fig. 3.—Brain of cat shown in Figures 1 and 2.

Fig. 4.—Cat two weeks after the removal of right lateral lobe of the cerebellum. Note its ability to support the body by its nonparetic limbs.

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Fig. 5.—Cat shown in Figure 4, after remaining in that posture for two to three minutes. Animal falls over to the right, owing to hyperactivity of the muscles supporting the body on that side (see text).

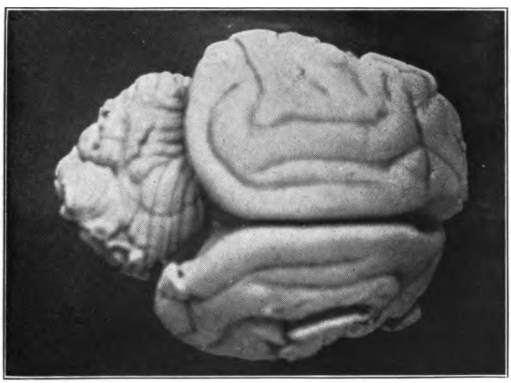

Fig. 6.—Brain of cat shown in Figures 4 and 5.

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on the other side, the former would be negative to the latter. That this is so for direct stimulation has been demonstrated by Hermann and Luchsinger, Waller and others in their study of skin currents. On connecting the pads of the hind paws of a cat with the galvanometer and stimulating the sciatic nerve for one of these, the corresponding pad has been found to be negative to the other. In this case the current in its internal circuit has to traverse, of course, from one side of the body to the other. That this holds true for indirect stimulation of tissue I have proved to my satisfaction by the following experiment:

With the cat under ether anesthesia, by means of a tracheal cannula I carefully exposed and isolated both sciatics. After drying all hemorrhage, I freed the nerves from contact with any of the surrounding tissue — which of course had been injured during the operation — and placed them on the boots of nonpolarizable boot electrodes, which had been soaked in Ringer's solution for the preceding twenty-four hours. I connected these with a galvanometer with a contact key in the circuit. The galvanometer employed during this and the other experiments to be described was of the Thomson type and manufactured by Elliot Brothers, London. It had a resistance of 20.74 ohms and its sensitiveness was such that at 37.5 cm. of scale distance 1 cm. deflection, registered 0.0000154 ampere of current, so that its constant (K) was 0.00155 ampere $\left(K = \frac{2\,I \times D}{d}\right)$.

While an experiment was in progress the nerves were kept moist with cotton sponges soaked in normal salt solution at a temperature of 38 C., which were removed during each observation so as to prevent any possible short-circuiting. The sponges were always applied to both nerves at the same time in order to avoid any possible differences of surface temperature. With the two sciatic nerves of the animal in the circuit as described, I made a number of observations on making and breaking the circuit, and was satisfied that they

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were equipotential, that is, that there was no current passing through the galvanometer. I then trephined the skull over the right motor area, both carotids having been ligated early in the operation, and exposed the cortex. The left sciatic promptly became negative to the right, the galvanometer registering each time the circuit was closed, a deflection of 1 or 2 cm., evidently as a result of irritation of the cortex by the exposure and the application of normal salt solution to its surface. The deflections then gradually diminished in range and were finally reduced to only 1 or 2 mm. I then stimulated the motor cortex by the faradic current and determined the area for flexion of the hind limb. Having fastened that limb securely to the board so as to prevent all movement of its muscles, I made observations of the galvanometer while the cortical motor area was being faradized. The left sciatic continued to be negative to the right, the deflection increasing to about 0.5 to 1 cm. I kept up these observations for about an hour and not once did the deflection take place in the opposite direction. I then trephined the skull over the left motor cortex and, as before, stimulated the hind-limb area there while the right hemisphere was at rest. The direction of the deflection was promptly changed, indicating that the right sciatic nerve became negative to the left. While these results were merely what was to be expected, their demonstration is of value in that it suggests to us the means whereby we are able to determine with more or less precision the comparative state of activity on the two sides of the body when one side is hyperfunctionating.

Applying this method toward the solution of our problem with reference to the mechanism of the cerebellar function, I carried out the following series of experiments: I removed in one group of cats the right lobe of the cerebellum; in another group the left lobe and kept them under observation for one, two and three weeks. I did not study them galvanometrically during

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the first week for two reasons. First, the animals were too much depressed and frequently in a state of collapse, and it was not thought wise to submit them to a second operation requiring prolonged anesthesia. Secondly, as my object was to determine the electric potential on the two sides, a state of negativity on the side of the lesion soon after the cerebellar operation would not indicate anything, as it might be due to irritation of the adjacent structures in the cerebellum. I therefore postponed such studies until all the irritative phenomena of Luciani had passed off. Out of all the animals operated on I selected for these studies only seven, three with the right lateral lobe removed and four with the left, as I discarded all those which did not show marked unilateral ataxia and whose wounds did not heal by first intention. Subsequent necropsies have shown that in each case almost an entire lateral lobe had been removed, leaving the vermis intact. The galvanometric studies were carried out in these animals in the same manner as in the experiment recorded above, only that in these animals no artificial stimulation was employed. The two ulnar or the two sciatic nerves were placed on the nonpolarizable electrodes which were in a circuit with the galvanometer, and observations made as to the presence or absence of a current and its direction. While the animals so studied were comparatively few, the results were so constant and so uniform for each kind of lesion that I feel justified in making certain deductions. The nerves on the side of the lesion, whether ulnar or sciatic, were invariably and persistently negative or "zincative" to those of the healthy side. The observations were kept up for two and three hours, and not once was there a deviation from this rule. Closing the circuit promptly brought about a deflection in the galvanometer, the direction of which indicated that a current was passing through it from the healthy side to the injured side. On changing, by means of a

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reverser, the poles of the circuit, the deflection, of course, took place in the opposite direction, always, however, indicating the same difference of potential between the two sides.

While the direction of the deflection was uniform, its extent was extremely variable, ranging from 2 cm. to 2 mm. within a very short space of time. This apparently depends on the condition of the animal and the state of narcosis, which, it should be remembered, virtually abolishes all nervous activity. That the anesthesia was a factor in determining the extent of the deflection was noted repeatedly when a deflection of only 2 or 3 mm. during deep narcosis became gradually increased on the withdrawal of anesthesia.

Could this difference in potential be due to differences in surface temperature of the two nerves, to differences in the concentration of the salt solution, or to other accidental causes? To determine this point I cut the nerves, leaving only the peripheral portions in contact with the electrodes. The deflections of the galvanometer ceased in each case, showing that the current previously observed was not due to such causes and also that it was not peripheral in origin. In carrying out this experiment care of course should be taken that the cross-sectional surface of the nerves is not in contact with the electrodes.

The question of course arises, what is the origin of this state of negativity or functional hyperactivity on the side of the lesion? It could not have been due to irritation of the cerebellar structures adjacent to the lesion as the same condition obtained three weeks after the cerebellar operation, when all possible irritative phenomena had passed off. It could not have been due to hyperactivity of the spinal cells on the side of the lesion, as, aside from the fact that these cells do not seem to possess any automatic activity, the spinal animal responding only to external stimuli, there was no evidence in my experiments of any hyperactivity of these cells. The reflexes were equal on both sides and

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there was no evidence of spasticity on the side of the lesion.

It may be assumed, however, that the hyperactivity originates in the paracerebellar nuclei, especially Deiters' nucleus. These nuclei, like the vagal cells in the medulla are known to possess automatic activity, as proved by the decerebrate rigidity following transection of the mesencephalon. Thiele[39] in an extensive study of this rigidity has shown that it originates in these nuclei and that the cerebellum exerts an inhibitory effect on them. Stimulation of the cerebellum produces immediate relaxation of such rigidity, as demonstrated by Sherrington[40] as well as Lowenthal and Horsley.[41]

It may also be assumed that the hyperactivity has its origin in the midbrain or the basal ganglia and that the inhibitory effect of the cerebellum is exerted on these structures. While later investigations tend to deny these structures a motor function, there is evidence that they participate in the production of movement to some extent.

And, finally, it may be supposed, as Gowers theoretically assumed, that there is hyperactivity on the side of the cerebellar lesion which originates in the contralateral motor area of the cerebellum.

To determine this point, I carried out the following experiments: In one cat I removed a very large portion of the right hemisphere of the cerebrum but left the motor area intact. In three others I removed only the motor area. The first cat showed no paralysis on the opposite side; the three others showed it to a very marked degree. Ten to fifteen days after the cerebral operation I removed in these cats the lateral lobe of the cerebellum, contralaterally to the previous lesions (of the three last, two had the right motor area of the cerebrum and the left lateral lobe of the cerebellum

40. Sherrington, C. S.: Proc. Roy. Soc. London, 1896, lx, 382; Integrative Action of the Nervous System, N. Y., 1906.
41. Lowenthal and Horsley: Proc. Roy. Soc. London, 1897, lxi, 20.

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removed, and one the left motor area of the cerebrum and the right lateral lobe of the cerebellum) and kept them under observation for fifteen days more. The first cat showed ataxia on the side of the cerebellar lesion, but, as before, no paralysis. The three last, on the other hand, were markedly paralyzed on that side. They showed no ataxia, but did show considerable tremor, involving head, trunk and paretic limbs. The presence of the tremor in these cases, after destruction of the motor area, would indicate that this symptom is not entirely cerebral in origin, a supposition advanced, on theoretical grounds, by Gordon Holmes[42] and Kinnier Wilson.[43]

Galvanometrically these cats gave the following results: The first one, with the motor area intact, although with a large portion of the cerebrum removed, showed a persistent and invariable negativity on the side of the cerebellar lesion, identically the same as in those animals in which a cerebellar lesion only had been produced. The three others, on the other hand, with a lateral lobe of the cerebellum removed and the additional extirpation of the contralateral motor area of the cerebrum, gave no galvanometric deflection whatever. I kept up my observations in these animals for nearly three hours and not once was there a deviation from this rule. Fearing that there was no deflection obtained in the last animals because of a possible fault in the circuit, I cut the sciatic nerve and applied the nonpolarizable electrodes to it in such a manner that one was in contact with the longitudinal surface and the other with the cross section. A current of injury was promptly obtained, showing that there was no such fault there.

The results obtained thus tend to show that the hyperactivity on the side of the cerebellar lesion, as

42. Holmes, Gordon: Brain, 1904-1905, xxvii, 364.
43. Wilson, S. A. Kinnier: The Anatomy and Physiology of the Corpus Striatum, Brain, 1914, xxxvi, 478; Progressive Lenticular Degeneration, Brain, 1911, xxxiv, 465.

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shown by its being electrically negative ("zincative") to the other side, originates in the contralateral motor area of the cerebrum; that following unilateral ablation of the cerebellum the contralateral motor area of the cerebrum becomes hyperexcitable, has been found by Risien Russell[9] and confirmed in part by Luciani.[4] Their results were denied by Bianchi.[44] Cerebellar incoordination is, therefore, in accord with Gowers' hypothesis, only indirectly cerebellar; it is primarily a cerebral effect, an expression of its hyperactivity consequent to the removal of inhibition or control by the opposite half of the cerebellum. The hyperactivity manifests itself in all the movements in that they are excessive, forcible, unchecked and go beyond the mark. In walking the animal throws its foot violently forward; in seizing food its paw goes beyond it, etc. Clinically, as Babinski pointed out, cerebellar patients, in performing the finger-to-nose test, are unable to stop the movement when the point aimed at is reached, and the finger goes beyond it, violently striking the jaw. Similarly, when tracing on a sheet of paper a line which should be stopped at a certain point, the pen goes way beyond this limit. The symptom of adiadochokinesia is therefore best explained by assuming it to be a result of the dysmetria and excess of each component movement, such as pronation and supination, a view suggested by Thomas, rather than that it is due to a delay in the excitomotor activity, as thought by Babinski, the discoverer of this phenomenon.

This conception of the cerebellar function is also in accord with certain well-established anatomic and clinical facts. The cerebellum develops embryologically from the rhomboidal lip of His, along the sensory tracts, as an accessory to them. Anatomically it is too, as Edinger, Sherrington, Gowers and others pointed out, a sensory organ. The effects of its lesions are, however, motor in character. This contradiction

44. Bianchi, quoted by Sherrington: Schäfer's Text-Book, ii, 904.

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between its anatomy and physiology does not exist if we assume the motor phenomena to be cerebral in origin, and the function of the cerebellum to be that of control and restraint, such, to use a crude simile, as that of the vagus, which is also a sensory structure.[45]

Another mystery cleared up by this conception of the cerebellar function is the path by which the cerebellar effects exhibit themselves on the periphery. As is well known, in view of the fact that the cerebellum has no descending tracts to the spinal cord, and also that the effects of its lesions manifest themselves homolaterally and not contralaterally, as is the case in lesions in all other motor and sensory tracts, it was found necessary to conceive an unusual and circuitous course for the transmission of its impulses. It is assumed that, originating in the deep nuclei of the cerebellum, the impulses first ascend by way of the cerebellomesencephalic tracts to the red nucleus of the opposite side and there are relayed, and finally descend by way of Monakow's bundle, or rubrospinal tract, crossing again to reach the spinal on the side whence they first started. This course, besides being different from that of all other motor and sensory structures which, as a rule, cross to the opposite side to exhibit their effect there, and, if homolateral in function, as the direct pyramidal tract or the vestibulospinal tract, go by direct route to the same side, is also untenable in the light of certain anatomic facts. The cerebellum attains its largest development in man, whereas the rubrospinal tract, by which its impulses are supposed to be carried, has been demonstrated only in animals and is in man at best only rudimentary.

According to our conception, however, each half of the cerebellum exhibits its function on the opposite hemisphere of the cerebrum, its tracts crossing to the other side like those of all other sensory tracts. Its

45. It was held by Waller, Schiff and others that the inhibitory fibers of the vagus originate in the nucleus of the spinal accessory. This seems to be disproved, however, by later investigations. (Luciani: Human Physiology, i, Chapter 9.)

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indirect effects, in the form of regulated and well-proportioned movements, manifest themselves on its own side, as, originating in the motor cortex of that hemisphere of the cerebrum, the motor impulses, passing by way of the pyramidal tracts cross to the opposite side before reaching the "final common path" in the spinal cord.

Finally, there is a good deal of evidence that there is a structural linkage between the cerebral hemisphere of one side and the cerebellar of the other side, and that the cerebellum is subservient to the cerebrum. It has been observed clinically in cases of congenital or acquired atrophy of the cerebrum, and experimentally after destruction of the motor cortex, especially if the optic thalamus was involved in the lesion, that there was simultaneously in the congenital lesions or after some time in the acquired variety, an atrophy or degeneration of the opposite lateral lobe of the cerebellum. These cases are described under the name of crossed hemiatrophy.

With reference to the tremor it appears to be largely dependent on an interaction between the cerebellum and the midbrain structures. I am engaged in some experiments with reference to these points at present and hope to communicate the results at some time in the future.

To sum up, the cerebellum is a complex structure having no direct effect on the periphery, but acting primarily on the motor cortex, the paracerebellar nuclei, and probably also the basal ganglia and ruber. Its primary effects are those of inhibiting, controlling and regulating the activity of these latter structures. Its ultimate effects are appropriate and rhythmic muscular action.

In conclusion I wish to express my indebtedness to Prof. A. J. Carlson of the University of Chicago for giving me the privilege and facilities for carrying out this work at his laboratories, as well as for a number of practical hints.

5 South Wabash Avenue.

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ABSTRACT OF DISCUSSION

ON PAPERS OF DRS. WEISENBERG AND WORK, GREY AND MEYERS

DR. W. F. SCHALLER, San Francisco: Dr. Work brought out the relationship between certain symptoms and definite localization of lesions. Certainly this is progress in the right direction. It is not a question only of posterior fossa disease characterized by a syndrome, it is a question of exact anatomic localization. The only way we shall make an advance in this work is by correlating the symptomatology and pathology; by studying our specimens in macroscopic and microscopic serial sections.

Dr. Meyers does not believe that nystagmus is primarily an expression of disturbed cerebellar function. I am inclined to favor this view, and regard vestibular nystagmus as a reflex and as the motor expression of a stimulus somewhere along the vestibulo-ocular path: labyrinth, vestibular nerve, Deiters' nucleus, posterior longitudinal bundle and oculomotor nuclei. This stimulus may be due to a focal lesion, or to pressure. Dr. Grey in his conclusions stated that in posterior fossa tumor without nystagmus he was inclined to believe that the tumor was intracerebellar; and that in cases of extracerebellar tumor he generally found nystagmus. May I ask Dr. Grey if the reason for this was not that in cases of extracerebellar tumor the intracranial pressure was increased and the nystagmus was due to disturbance of function by pressure? If there be a nystagmus following cerebellar lesions proper, I believe it is due to involvement of the vermis in relation to its efferent fibers to Deiters', i. e., cerebellovestibular tract (*Hacken Bundel, faisceau en crochet*) which forms an arm of the nystagmus reflex in that stimuli are received in this way from the muscles and joints and pass to the vermis before acting on Deiters' nucleus. To illustrate these views I wish briefly to mention three posterior fossa cases recently studied clinically and anatomically. In the first case there was no nystagmus, no intracranial pressure. The lesion was a vascular one, limited to the white matter of both cerebellar hemispheres and involving the dentate nucleus on one side. There was marked ataxia of the cerebellar type. The vestibulo-ocular reflex path was not involved; hence absence of nystagmus. The second case was that of a right sided cerebellopontine angle tumor in which the usual signs of intracranial pressure, vertigo, nausea, headache and choked disks were absent. The tumor had caused a considerable atrophy of the cerebellar hemisphere on the same side and also the pons, and by replacing these structures had not materially added to the contents of the posterior fossa; hence absence of intracranial pressure. Ataxia of cerebellar type was marked. A slight nystagmus more marked to the right was explained by direct involvement of the vestibulo-ocular

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reflex. The third case was a tumor in the fourth ventricle arising from the inferior tela. The marked nystagmus could be explained by direct pressure of the growth on Deiters' nucleus. Ataxia was absent.

Dr. A. L. Skoog, Kansas City: The subject of cerebellar tumor is always interesting, and might be broadened to include any of the lesions in the cerebellum. The subject of cerebellar function has received a great deal of attention in the last few years and many new facts have been presented. However, as indicated by Dr. Work, many of the statements are really theoretical, which perhaps makes the subject still more interesting. As stated by several of the speakers, it invites the possibility of error in diagnosis. It has been stated that glioma is the most frequent type of tumor found in the cerebellum. This is in accordance with my own view. The glioma is a type of tumor which always springs from glia tissue, but there are those gliomas which are more dense than others, those that have a tendency to be better outlined. The type, however, is the infiltrating one and at times they are rather difficult to differentiate from some simple degenerating conditions of the central nervous tissue. I have always been interested to know whether there is any difference in symptomatology, even though the tumor be located in the same place, between the soft and infiltrating and the more dense one. That might be a reason for the difference in symptomatology which was brought out in the discussion.

Dr. G. W. Robinson, Kansas City: Dr. Work speaks of the diagnosis of tumors in the posterior cranial fossa. I should like to ask Dr. Work to speak on the differentiation between these and those situated more anteriorly and giving cerebellar symptoms. I recall two or three patients with lesions (tumor or trauma) of the frontal lobe, chiefly of the middle frontal convolutions, the apraxic center, giving a contralateral cerebellar syndrome. Another condition I have observed recently has been a patient with chronic progressive cerebellar tremor, without ataxia or nystagmus. I should like some of the men who read these excellent papers to tell us, if possible, the location of the lesion giving this particular symptom. I have recently observed two patients who had shown no nystagmus. At necropsy one had a degeneration of the right cerebellar hemisphere resulting from an encephalitis (associated with typhoid fever). The other had a large cyst involving the right cerebellar hemisphere.

Dr. John S. Turner, Dallas, Texas: These cases of cerebellar disturbance are very interesting to all of us and I rise to report a case and to present a question and to ask some of the gentlemen in closing to touch on it. It is in reference to a case recently occurring in our part of the

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country which became almost a medicolegal case, but by having the records properly presented a damage suit was prevented. The case came under my observation with all the typical symptoms of cerebellar tumor—nystagmus and other eye symptoms. The patient was a girl of 18, being able to go about, but not seeing very well, and having headaches, vomiting, titubation, disturbed coordination and considerable emaciation running over a long period of time. After observing the symptoms and making a tentative diagnosis, I sent her to a physician to look over the eye conditions. He confirmed my diagnosis in so far as the eyes were concerned, using some atropin to further dilate the pupil. Immediately after the dilatation the patient became blind and has never seen since that moment. Her headaches ceased, she gained in weight and was physically much better, but was totally blind. A damage suit was threatened against the physician for making the patient blind, but the records showing that she was partially so, and the opinion that she would ultimately become blind anyway prevented the suit.

Dr. Cecil E. Reynolds, Los Angeles: Dr. Work laid stress on the paralysis of the sixth nerve in cerebellar lesions, and I think it is very important. I remember a case of Dr. Charles A. Ballance of London at the Great Ormond Street Hospital in 1905, in which there was weakness of the sixth nerve on the left side and a bulging of the occipital fossa on the right side. The optic neuritis was more advanced on the left side. Mr. Ballance operated on the side which bulged, but the tumor was eventually discovered on the same side as the sixth nerve paresis. In my experience the signs of intracranial pressure have been more intense in cerebellar lesions and more early than in cerebral lesions, and this fact in a measure militates against the value of sixth nerve paralysis as a localizing sign in cerebellar disease, because in general increase of intracranial pressure the sixth nerve is often first affected by reason of that general pressure.

I would like to ask some of the gentlemen who have spoken as to their experience of the value of skew-deviation as a localizing sign. Dr. Robinson mentioned a confusion of frontal lobe lesions with cerebellar lesions. I have seen several cases in which a frontal lobe lesion was mistaken for a cerebellar lesion, and it appears to me that in cerebellar lesions the head is more twisted, with the chin pointing to the shoulder of the affected side, whereas in frontal lobe lesions the eyes deviate to one side or the other without much tilting of the head. Also I have noticed that in addition to hebetude out of proportion to the pressure, there is usually a slow and deliberate Babinski's sign in frontal lobe lesions, more sluggish than that usually seen in lesions of the pyramidal tract.

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DR. GEORGE A. MOLEEN, Denver: Just one remark on what Dr. Work said with reference to the extension of tumors from the thalamus downward. Is it not the rule to find unilateral paralysis of the third nerve prior to the involvement of conjugate deviation, the center of which lies pretty close to the sixth center in the pons? From this we presume there is a communicating fiber to the third, and in these extending tumors it would seem fair to expect a unilateral lesion of the third before the affection or disturbance of conjugate deviation would occur.

DR. PHILIP WORK, Pueblo, Colo.: We found in working up our pathologic material on this subject that cases which we thought definitely cases or tumor of one or other peduncle proved to be cases in which the involvement had gone past the peduncle, and the diagnosis and localization of symptoms has been hindered by the fact that these cases are so far advanced before they come to section. I personally believe that the matter of function of the peduncles—and coincident with it the establishment of a definite symptomatology —can only be hoped for when we have seen a sufficient number of early cases come to section. This is predicated on a series of necropsies from coincident cases in which the brain lesion was discovered incidentally in routine examination. The differential diagnosis of cerebellar and frontal tumor has been mentioned by one gentleman, but is too big a subject, I feel, to take up here. I have in mind several cases in our own series in which that identical question came up. In two cases in which the clinical diagnosis was made of cerebellar trouble, section showed frontal trouble, with definite cerebellar symptoms. These cases are being worked up at the present time, and we hope to be able to report something of value when we get them correlated. The loss of sight spoken of in the medicolegal case would seem to me to be due to the advanced state of the optic condition. The fact that the other symptoms abated more or less I do not attempt to reconcile.

DR. ERNEST GREY, Boston: Dr. Schaller has referred to increased intracranial pressure as an important factor in the production of nystagmus. This is the view of Bárány and others, and in our earlier work we inclined to the same belief. Recent observations, however, have led us to seek another explanation. We found nystagmus present in a group of patients with subtentorial new growths, some of whom had greatly increased intracranial pressure, while others had practically normal pressure. We have had a similar series of patients with the same variations in the degree of intracranial tension; none showed any nystagmus.

DR. I. LEON MEYERS, Chicago: Something has been mentioned in regard to nystagmus. I can simply add what I said in my paper—in my experience true cerebellar lesions

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do not give rise to nystagmus unless there is additional involvement of the vestibular complex. Skew-deviation has been observed in a few experiments on dogs during the time of anesthesia, but was lost after the animal awakened. Frederick Batten in a paper published in *Brain* in 1903 determined that the position of the head is not diagnostic of cerebellar lesions. It appears that the position of the head is due to the loss of knowledge of special relationship and is vestibular in origin. In my judgment the cerebellum is purely an organ which exerts an influence on other nerve cells in the brain structures—not on the periphery; the control it exerts is chiefly in the motor sphere.

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ACUTE ASCENDING HEMORRHAGIC MYELITIS

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The title of this article is descriptive of a rare pathologic condition which has comparatively recently come under my observation. Its rarity is attested by the fact that in the literature very few cases can be discovered of this combination of acute inflammation of the spinal cord with overwhelming diffuse hemorrhages into the gray matter, and to somewhat less extent into the white matter.

It seems important to draw an analogy between this case and two other cases of acute ascending paralysis, also recently under observation.

The evidence tends to show that there are three clinical as well as pathologic types of acute ascending paralysis. My conception of the differentiation of the three will follow later.

In order to have the material before us, I shall at once describe this case of acute hemorrhagic myelitis, and later briefly the other two cases.

CASE 1.—E. W., a girl of 20, about to graduate from a private school, gave the following history: Her parents are living and well. There are two other children, one well and the other nervous. No family disease is known.

The patient was a "blue baby," and the blueness persisted throughout her life. She had been rather delicate, of a nervous type and had had to favor herself more than a normal child. She had done well in school, however, and up to five days ago appeared to be in her normal health. Five days ago she first noticed a numbness, accompanied by some weakness, in her left foot. The following day a similar

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condition arose in the right foot, and later this numbness and paralysis gradually crept up her body, so that on the third day the numbness extended as far as the chest and began to attack the left arm.

She was brought home at this time, and was attended by two physicians, who were undecided whether she was suffering from hysteria or from some spinal cord lesion.

On the next day she had for several hours very severe pains, starting apparently in the spinal region and extending down both legs, especially the left. About this time also, her right arm became involved, the neck became somewhat stiff and she was practically completely paralyzed, except for her head, though certain weak movements of the right hand were possible.

At this time, that is, on the fifth day, I saw her in consultation with Dr. Kendall Emerson, and made the diagnosis of acute ascending paralysis. The temperature at this time was 103. The whole body was a grayish blue. The finger tips were markedly clubbed. There was practically complete anesthesia and muscular paralysis below the neck. The patient was unable to control the bladder and rectum. On the left leg was a large burn, 6 inches across, of which she was not aware, produced the night before by a hot water bottle. The pupils were equal and reacted normally to light. The neck was only slightly rigid. The tongue was dry and coated. The heart was rapid, 120, but not markedly enlarged and no murmurs were heard. The patient was conscious and perfectly clear mentally. The speech was unaffected. The respiratory muscles were just becoming affected, deep respiration absent. No glands were palpable.

I ordered immediate removal to Memorial Hospital, after first doing a lumbar puncture and withdrawing 15 c.c. of a somewhat turbid and markedly blood-stained fluid. The pressure of the fluid was somewhat above normal. The blood stain was evidently not due to trauma by the puncture, as all the fluid withdrawn was of the same nature. Subsequent examination of this fluid showed a marked excess of blood cells, an enormous excess of polynuclear white cells, some of them more or less disintegrated, and no bacteria. Cultures were taken from the fluid but no organisms could be grown. The globulin reaction was positive.

At the hospital the patient was made as comfortable as possible. She had no pain, but felt as if her body was heavy and she had an increasing sense of exhaustion. She was put on liquids and hexamethylenamin, 20 grains, every four hours.

Her entrance to the hospital was at 8 p. m., May 25, 1914. At that time her white blood count was 12,300; hemoglobin 80 per cent.

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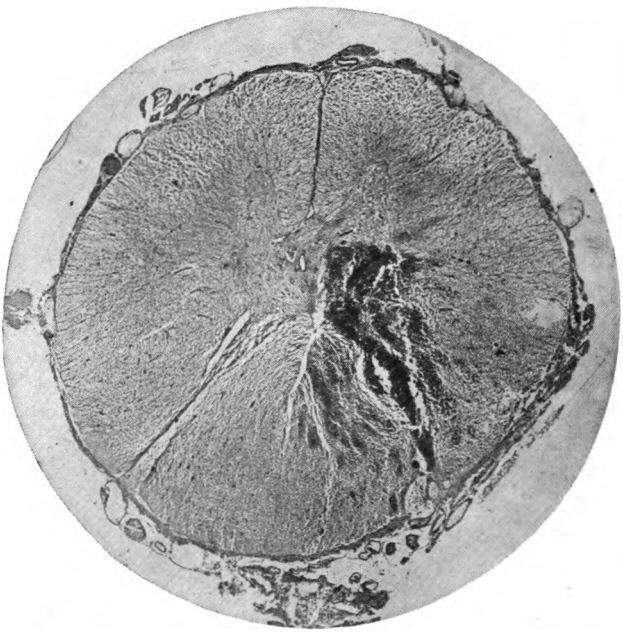

Fig. 1.—Case 1, E. W. Cord showing large area of hemorrhage in posterior horn.

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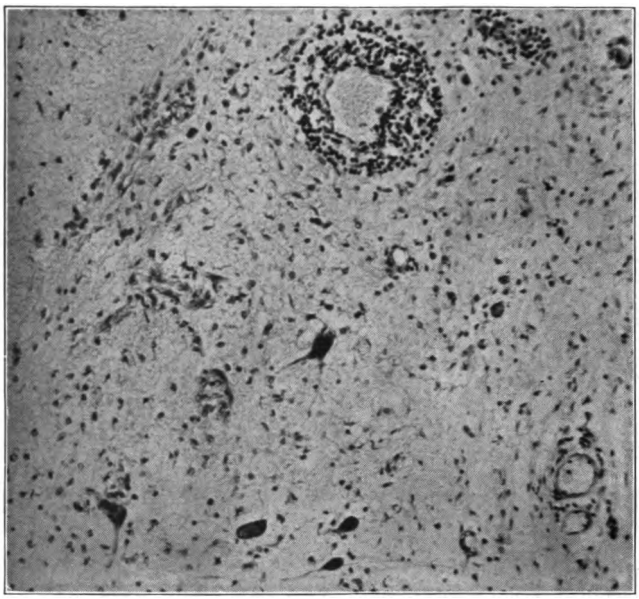

Fig. 2.—Case 1, E. W. Dorsal cord. An area from the anterior horn, showing lymphocytic perivascular infiltration. The nerve cells are intact.

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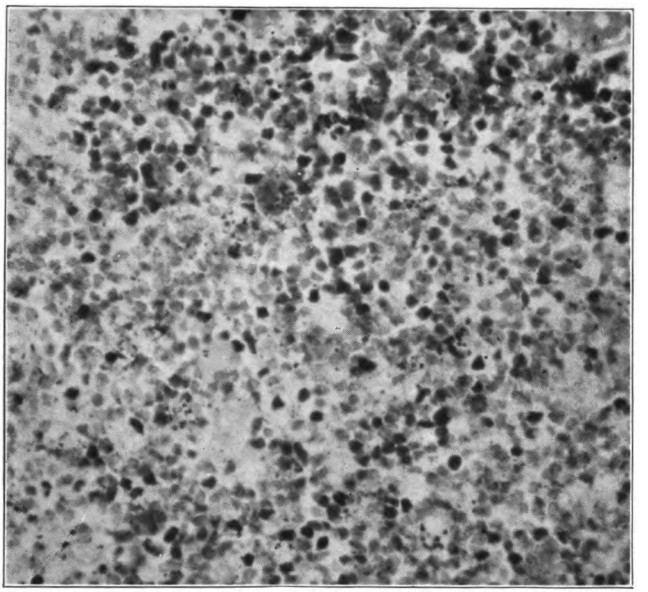

Fig. 3.—Case 1, E. W. Area of hemorrhage in the gray matter.

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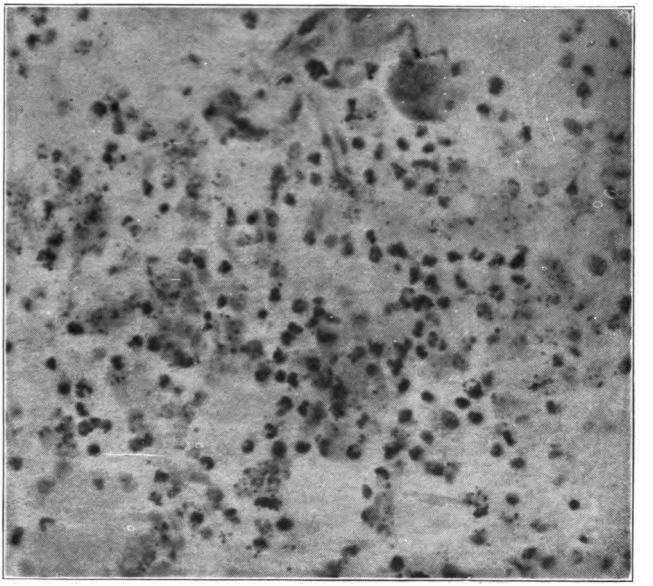

Fig. 4.—Case 1, E. W. Exudate, showing the large proportion of polymorphonuclear leukocytes, beginning necrosis and the presence of blood pigment.

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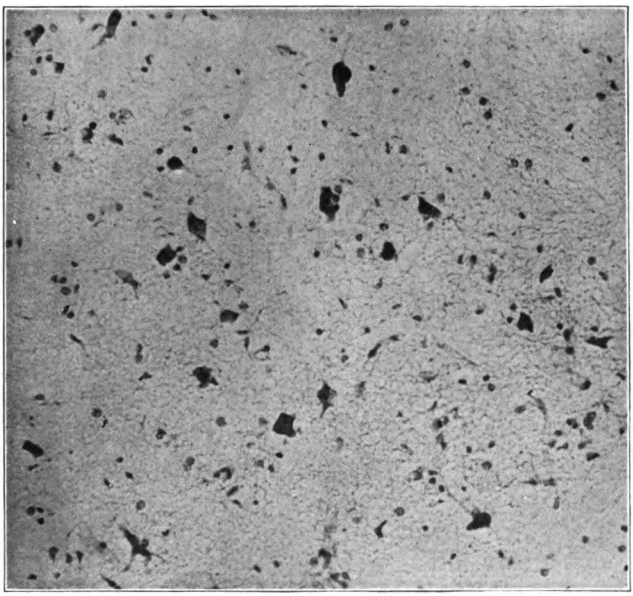

Fig. 5. — Case 2, E. C. Nucleus of VII, showing degeneration of the nerve cells and the presence of satellites.

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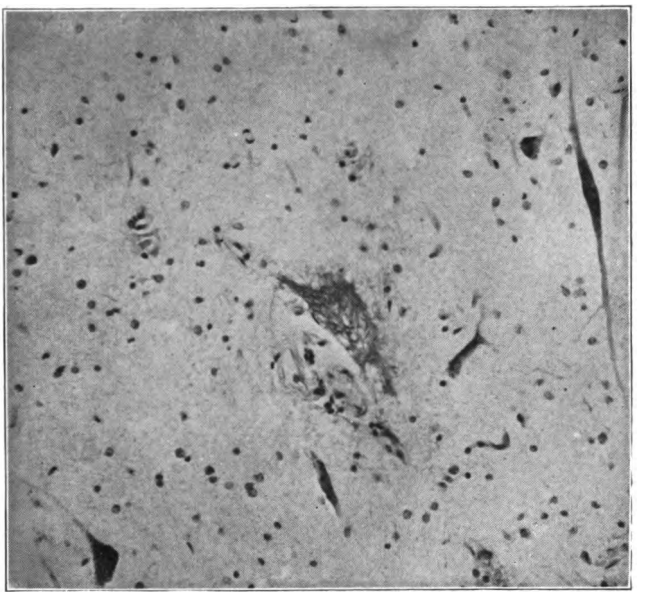

Fig. 6.—Case 2, E. C. Lumbar cord, showing cells of the anterior horn. Small numbers of lymphocytes and an area of fibrin network about a vessel. A degenerated cell on each side of the vessel.

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She did not sleep well that night on account of inability to move and a sense of weakness. Toward morning the respiration became more shallow and cyanosis increased. Suddenly at 9:45 a. m. respiration stopped practically without warning. A few moments later the heart beats ceased.

Permission was given for necropsy, which was performed by Dr. Kinnicutt at 1:30 p. m.

Anatomic Data.—Body of a well-developed young woman, markedly cyanosed.

Head: On section of the scalp there is a large amount of dark fluid blood. On removal of the skull-cap the longitudinal sinus is distended with dark fluid blood. The dura is not adherent, the meningeal vessels are injected, and the convolutions are somewhat flattened. On section of the brain: The ventricles contain no excess of fluid, but throughout the cerebral cortex are scattered numerous minute hemorrhagic areas.

Spinal Cord: The sections throughout its length show extensive areas of hemorrhage within the cord, especially into the gray matter, often entirely replacing the normal markings. There is no excess of spinal fluid and no evident induration around the meningeal covering.

Body: On section the subcutaneous fat is considerable in amount. The peritoneum is smooth and glistening. The appendix is normal. The lower border of the liver in the right mammary line is at the costal margin. The pleural cavities contain no free fluid. The lungs are salmon red in color and on section yield a large amount of pale, frothy fluid. The pericardium is normal. The heart is somewhat enlarged. On section the left ventricle wall is thickened. The papillary muscles are hypertrophied. The valves are normal. The foramen ovale is patent. The liver on section yields a large amount of dark fluid blood and shows "nutmeg" markings. The spleen is enlarged, firm, dark purple in color and on section yields a large amount of dark fluid blood. There is a small accessory spleen at its lower pole. The kidneys are enlarged. The capsules peel easily, leaving smooth surfaces. The abdominal organs otherwise are not remarkable.

Sections of the brain and spinal cord are placed in alcohol or 10 per cent. dilution of liquor formaldehydi for further examination. The bulk of the spinal cord is put into sterile glycerin for shipment to the Rockefeller Institute for Medical Research.

Microscopic Examination of the Brain and Cord: Paraffin Cresyl Violet Sections.—Cortex: Section 1: Precentral. The pia is of normal thickness. It contains small numbers of lymphocytes and numerous large endothelial cells, many of which are laden with blood pigment. Cortical lamination is normal and the nerve cells stain well. A mild satellite

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reaction is present in the deeper layers. Small amounts of green pigment are seen about some of the cortical capillaries, but otherwise the vessels are normal.

Section 2: Location unidentified. The meshes of the pia are filled with red corpuscles together with moderate numbers of lymphocytes and large mononuclears. A marked satellite reaction is present in the infrastellate layers of the cortex, some of the cells resembling neurophages. Some of the nerve cells which are surrounded by satellites are shrunken, and stain deeply and diffusely. Around several vessels in the white matter are collections of lymphocytes and pigmented phagocytes.

Thalamus: Ependyma is normal. Considerable satellitosis. Small collections of lymphocytes and green pigment about some of the vessels.

Cerebellum: Pia contains a moderate infiltrate of large mononuclears with a scattering of polymorphonuclear leukocytes, lymphocytes and red corpuscles. The mononuclears are extensively phagocytic, being stuffed with lymphocytes, polymorphonuclears, red corpuscles and pigment. Cerebellar tissue is not remarkable.

Cord: Section 1: About second segment (Fig. 1). Macroscopically, a large area of hemorrhage is seen, occupying the anterior and posterior horns of one side and extending anterolaterally to the periphery. Microscopically, there is found a disruption of the nerve tissue in the hemorrhagic area and a marked edema of that side of the cord. Smaller hemorrhages are scattered about the large focus. The reaction of the nerve cells varies. An occasional one is shrunken and angular, but the majority stain well, even those situated in the hemorrhagic area. There is no undue satellitosis. A heavy perivascular infiltrate of lymphocytes, together with a few polymorphonuclear leukocytes, is present. No plasma cells are seen. The vascular endothelium is not remarkable. Numerous glia cells with large vesicular nuclei and visible cytoplasm are present in both gray and white matter. In the central part of the hemorrhagic focus are large numbers of polymorphonuclear leukocytes and areas in which the tissue stains a dull, diffuse blue, suggesting necrosis. The opposite side of the cord shows a small area of hemorrhage, considerable edema of all the structures, and a mild lymphocytic perivascular exudate.

The pia is comparatively unaffected by the process, showing only occasional lymphocytes about the vessels.

Section 2: Lower cervical. Changes in the cord are similar to those in Section 1. There is a mild lymphocytic infiltration about the branch of the anterior spinal artery in the anterior fissure, and scattered lymphocytes among the anterior and posterior root bundles.

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Section 3.—Lower dorsal. Very marked edema throughout the cord, which, with several large areas of hemorrhage, distorts the outline of the gray matter. Perivascular exudate as in Section 1. There are collections of lymphocytes, polymorphonuclears and large mononuclear cells in the pia about the points of exit of the anterior roots. There is no exudate, however, among the nerve bundles themselves.

Alzheimer's Lichtgrün Stain.—Cortex: Large granules staining with osmic acid are found rather abundantly in the cells in the perivascular spaces of the white matter, and to a small extent in the vascular endothelium of both cortex and white matter, and in a few nerve cells. No ameboid glia.

Lumbar Cord: Very heavy black deposits in the nerve sheaths everywhere in the white matter and in the nerve roots. Numerous glia cells with large vesicular nuclei and ameboid bodies are present in the white matter. These cells frequently contain vacuoles or black granules. The cord is infiltrated with hemorrhage.

Mallory's Phosphotungstic Acid Hematoxylin Stain.—No increase of fibrillar glia in cerebrum, cerebellum or cord. Examination of numerous cresyl-violet, thionin and Zenker-eosin-methylene blue sections reveals no bacteria.

Summary.—An acute diffuse inflammation of the cord, characterized by extensive hemorrhage, heavy perivascular infiltration of lymphocytes, combined with smaller numbers of polymorphonuclear leukocytes, ameboid glia and fatty degeneration of the fibers in the cord and nerve roots. The nerve cells are in general well preserved, and there is no neurophagic activity. The centers of the large hemorrhagic foci show beginning necrosis with large numbers of polymorphonuclears.

The cortex shows a mild meningeal lymphocytic reaction, small deposits of lipoid in nerve cells and vascular endothelium, occasional perivascular lymphocytes and phagocytes, and in one section a marked satellite reaction with slight degenerative changes in the nerve cells.

As I intimated in the beginning, little success has been met in the search for cases like the one just reported. Of course a number of cases of anterior poliomyelitis have been reported with more or less hemorrhage into the gray matter, also many cases of more or less diffused myelitis, with hemorrhage. None of these, however, seem to correspond exactly to my case.

Wyss reported before the International Congress in 1898 a case of hemorrhagic myelitis and phlebo-

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thrombosis of the spinal cord, in a girl of 12 years. This process, however, was found to be dependent on a tumor of the cervical cord, and pathologic lesions did not extend above this area.

Warrington and Owen have since reported a case somewhat similar. The history is as follows:

A man 41 years old. Illness began with chill, and three days later he had a temperature of 101. He then seemed to improve, but four days later he developed sensory and motor paralysis of both legs, with sphincter involvement. The paralysis ascended during the next four days until the respiratory center failed.

At necropsy the cord at various levels showed masses of intraspinal hemorrhage, more severe in the dorsal region, and corresponding chiefly to the anterior spinal system of arteries. A distinctive feature was the great dilatation of the small vessels, in both gray and white matter. The pia also showed greatly distended vessels and cell extravasation. Under high power the vessels showed wall thickening and neighboring necrosis of tissue. The cornual cells and nerve fibers exhibited all degrees of degeneration when near the vascular disturbance. Beyond these zones the nerve structures were intact. The brain above the medulla was normal. No micro-organisms could be detected.

This case, though dissimilar, is more like Case 1 than any other I have found. By way of comparison, let us now consider Case 2.

CASE 2.—E. C., a young man of 26, occupation chemist, gives no abnormal family history. Aside from mumps and scarlet fever in childhood and a rather prolonged cough one year ago he has been well. He graduated from college four years ago and has since then followed his occupation.

Present Illness.—Patient has been working hard in a rubber mill without a vacation and has felt somewhat tired for a month or two. Three weeks ago he began having headaches which were not constant but have become much worse within the last three days. Four days ago he complained of numbness in both thighs and legs and they did not seem as strong or steady as usual. Two days ago the patient's gait became more unsteady and he took a long walk trying to get the stiffness out of his legs. This, however, only had the result of tiring him further and caused considerable perspiration and general weakness. Yesterday it was noticed that the lines were lacking in the left side of the face and there was some drooping of the left eyelid with loss of power

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to close the lids. The numbness has extended up through the thighs and there has been a slight numbness in the tongue, especially of the left side. No sphincter disturbance at time of consultation with Dr. Prouty of North Brookfield.

Physical Examination.—July 16, 1913. Patient is fairly well nourished. Skin moist. Pupils are equal and react to light and accommodation. Slight exophthalmos. Left facial paralysis. Slight difficulty in speech. Heart and lungs negative. Extremities: Knee jerks absent. Plantar and Achilles reflexes absent. No Oppenheim or Gordon present. Some ataxia. Romberg suggestive. Coordination of arms fairly good. General weakness of legs. Sensation: Tactile, temperature, pain and muscle sensations are well preserved all over the body, though the legs have peculiar sensations to touch. Tache cérébrale is marked.

Patient was sent directly to Memorial Hospital and two hours later lumbar puncture was done with withdrawal of 15 c.c. of spinal fluid.

Spinal Fluid Examination: Pressure normal, fluid clear, globulin positive, Fehling's reduced. Cell count 14, practically all lymphocytes (Alzheimer).

Course.—July 17: Weakness of legs has become so marked that patient cannot stand on them. They are moved weakly in bed, however. Intense pain in the head and neck is complained of, only partially relieved by ice and migraine tablets. Fundus examination by Dr. Harrower with negative report.

During the next two days the paralysis gradually extended upward to the muscles of the abdomen and a partial facial paralysis developed on the right side. The patient was very restless, but the headache was possibly less severe than at first. Subjective numbness was marked in the legs.

Two days later it had become extremely difficult for the patient to turn in bed. An involuntary defecation occurred. The respirations became shallow and the voice not strong. Restlessness continued in spite of medication.

On account of the evident involvement of both seventh nerve nuclei, I requested Dr. Lovell to test the hearing, especially for oversensitiveness, suspecting involvement also of the eighth nerve. The report, however, was negative.

July 23: One week after entering the hospital, the paralysis had extended to both arms and they were somewhat prickly but not anesthetic. Only liquids could be swallowed readily. Sphincters of bladder and rectum disturbed.

The picture of paralysis was not particularly changed during the next three days; in fact, there appeared to be less involvement, especially of the seventh nerve, and rather less paresthesia of the legs. The respiration, however, was very shallow and the pulse rate increased.

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July 26: At 5 p. m. the patient, on trying to take a Seidlitz powder, choked suddenly and respiration ceased. Artificial respiration was tried and, a few moments later, a pulmotor was used, but the heart had failed and the patient expired.

The temperature had hardly reached 99 at any time, and the pulse rate varied from 80 early in the illness to 120 toward the end.

Urinary examination showed no abnormality except the slightest traces of albumin. The medical treatment consisted chiefly of fairly large doses of hexamethylenamin early in the illness, with salt solution by rectum and subcutaneously in the later stages.

Blood Examination: Hemoglobin, 90 per cent.; leukocytes, 11,100; erythrocytes, 5,628,000. Differential: Polymorphonuclears, 71 per cent.; lymphocytes, 21 per cent.; large mononuclears, 6 per cent.; mast cells, 1 per cent.; eosinophils, 1 per cent.

Necropsy.—By Dr. Hunt, three hours after patient's death. Nothing remarkable about the viscera of the chest and abdomen except that the distal part of the ileum was considerably injected and there appeared to be a general enlargement of the intestinal lymph nodes. Many of these nodes were the size of a cranberry bean and firm to pressure. The kidneys were slightly enlarged, but had normal markings and freely removable capsule.

The spleen was slightly enlarged and showed some signs of congestion. Cultures taken from the heart and subdural space failed to produce any organisms. On removal of the calvarium an excess of edema and considerable vascular congestion was noted in the meninges. The arachnoid was glistening and appeared somewhat less transparent than normal. The meninges of the cord likewise gave the appearance of congestion and edema.

On fresh section of the cord and brain no gross pathologic lesions were evident.

It was planned to inject emulsions of spinal cord from this case into monkeys to help establish the etiology, but, on account of the absence of the laboratory staff from two hospitals, it failed of accomplishment.

Macroscopic Appearances.—Brain: The pia appears somewhat glazed, but is delicate and strips easily. The convolutions are broad and rather simple in pattern, but well filled out. No gross lesions found on cutting the brain.

Basal Vessels: Delicate.

Spinal Cord: Pia shows some injection of vessels. No macroscopic lesion.

Microscopic Examination.—Paraffin Cresyl Violet Sections.—Precentral Cortex: Pia is of normal thickness and contains small numbers of lymphocytes and pigmented phagocytes.

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Nerve cells are present in apparently normal numbers and arrangement, but many stain poorly, being shrunken and angular, with visible processes, darkly and diffusely staining cytoplasm and pyknotic nuclei. A few cells are reduced to shadows. A number of nerve cells, however, stain well and show a good content of Nissl granules. The giant pyramids are the best preserved of the nerve cells and appear entirely normal. A mild satellitosis is present. The vessels are of normal thickness. The perivascular spaces contain scattered lymphocytes and considerable amounts of dark green pigment.

Postcentral: Resembles precentral.

Frontal: Resembles precentral. Abundant perivascular collections of green pigment.

Temporal: Resembles precentral.

Occipital: The cells stain better than in the preceding cortices. Slightly increased satellitosis in the deeper layers. Perivascular pigment is small in amount.

Hippocampus: Not remarkable.

Cerebellum: Pia is similar to that over the cerebral cortices. Purkinje cells are present in normal numbers and stain fairly well. Small collections of green pigment and lymphocytes about the vessels in the central white matter. The dentate nuclei contains quite a number of shrunken cells with opaque, eccentric nuclei.

Thalamus: The nerve cells stain normally. No increased satellitosis or perivascular changes.

Pons: Showing the nucleus of seventh nerve. In some groups of this nucleus the nerve cells stain excellently, in others there are numerous degenerate cells, some of which are surrounded by satellites (Fig. 5). Numerous cells in the nucleus of the sixth nerve are also affected. A few withered cells are found in the nuclei pontis. A nerve trunk (probably sixth) at the lower edge of the section shows a few lymphocytes around its vessels, but nothing very marked. Pial changes as over cerebrum.

Medulla: At the point of entrance of the seventh nerve. Scattered degenerated cells are found in the dorsal and ventral nuclei of the cochlear division. Isolated, degenerated cells are also present in the nucleus ambiguus and the olivary nuclei. There is considerable satellite reaction around the degenerated nerve cells, some of the satellites resembling neurophages. Small collections of lymphocytes are seen about a few of the vessels in both the nerve tissue and pia. The vessel walls appear normal. There are also a few lymphocytes about the vessels of the eighth nerve, but no infiltration along the nerve bundles.

Medulla: At the level of the lower part of the corpus restiforme. The cells in the nucleus of the twelfth nerve are well preserved and have a full content of Nissl bodies.

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The dorsal nucleus of the tenth nerve forms a sharp contrast to the twelfth, showing many shrunken cells. Similar degenerated cells are scattered through the nucleus ambiguus and a few cells showing the axonal reaction are present. Perivascular conditions as in preceding section. The ependyma is normal.

Medulla: At level of decussation of pyramids. The pia shows a sprinkling of lymphocytes about the vessels. Some of the cells of the anterior horns stain normally, but scattered among these are others which are shrunken and angular, have visible processes and dislocated nuclei and stain deeply and diffusely. Similar abnormal cells are found in the nuclei of the columns of Goll and Burdach. The small cells in the ascending root of the fifth nerve appear normal, while many of the larger are affected. There is no perivascular exudate.

Cervical Cord: About the fourth segment. The cells of the anterior mesial group are normal. In the group posterior to that, however, which probably represents the nucleus of the phrenic nerve, are numerous cells showing degenerative changes. Some of these cells are surrounded by satellites. Pial changes are of the same grade as over the cerebrum, and there is very slight perivascular lymphocytosis in the gray matter. The white matter is not remarkable.

Dorsal Cord: About the sixth segment. Scattered nerve cells showing degenerative changes are found in the anterior and posterior horns and in Clarke's column. Most of the cells, however, stain very well. There are scattered lymphocytes about a few vessels in the cord, but the meninges are entirely free from exudate. The posterior root bundles included in the section show no changes.

Lumbar Cord: First segment. There are collections of lymphocytes with an occasional pigmented phagocyte in the anterior fissure, but otherwise the meninges appear normal. The nerve cells are as above. At the side of one perivascular space and extending into the tissue is an area of fibrin network (Fig. 6).

Lumbar Cord: Second segment. The cells of the anterior mesial group stain well, but moderate numbers of degenerated cells are found in the lateral groups and especially among the small nerve cells of the posterior horns. There are scattered lymphocytes about the vessels of the anterior and posterior nerve roots, but no perivascular exudate in the cord.

Lumbar Cord: Fifth segment. Scattered degenerated cells are present in all groups, but the majority of the nerve cells stain well. There is a scanty lymphocytic infiltration about the vessels to the anterior horns. The pia and the anterior and posterior roots are not remarkable.

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Phosphotungstic Acid Hematoxylin Stain for Neuroglia.—Sections of the various cortical regions and the cerebellum show no superficial or deep gliosis. Lymphocytes are quite abundant in the pia, particularly at the bases of the sulci. Perivascular pigment is well brought out by this stain. It is more abundant in the central and frontal regions, and more prominent in the subcortical white matter than in the cortex.

Examination of the cord in the cervical, dorsal and lumbar regions by this method gives no additional information.

Marchi Stain.—The pia and the perivascular spaces of the cortex contain abundant coarse brown granules. The nerve cells show moderate deposits of similar granules and the neuroglia cells a few. Small numbers of black droplets are scattered in the white matter.

Cord: No Marchi sections.

Alzheimer's Licht-Grün Stain.—No glia cells of the ameboid type. The lipoid deposits are well brought out.

Weigert Stain.—Sections are not notable.

Summary.—A widespread degenerative process in the nerve cells of the cerebrum, cerebellum, medulla and cord. The affected cells are frequently surrounded by others which appear normal, and no particular groups can be said to be peculiarly involved. There is a moderate satellite reaction, moderate perivascular lipoid deposits, and slight perivascular lymphocytosis in both pia and nerve tissue. The nerve roots, both cranial and spinal show small lymphocytic deposits.

The pathologic picture is one which has been frequently described in cases of Landry's paralysis, that is, a widespread degeneration of the nerve cells with scanty perivascular exudate. The process in the present case is one which strikes the nerve cells differentially. Some fade out with, others without satellitosis. The picture is compatible with a purely toxic condition.

Further discussion of this case is postponed until after a brief summary of the following case.

CASE 3.—A boy, 14 years old, was always well until Sept. 13, 1913, except for an accident to his head a year previous, at which time he received tetanus antitoxin. Two weeks previous to September 13, he had returned from a vacation at the shore in excellent health.

The early symptoms noted were headache and fever, which, on the afternoon of the 13th, reached 102.5. The headache, fever and malaise continued the next day, and during the night of the 14th paralysis of both legs developed. This paralysis gradually ascended during the next thirty hours until the thighs, abdomen and arms and respiratory muscles

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became in sequence involved. No pain was complained of. There was some paresthesia, but no actual anesthesia of the limbs. The deep reflexes of both legs and arms were absent. The paralysis was flaccid. Lumbar puncture was done by me at this time, September 16, on being called in by the family physician.

The spinal fluid was under moderate pressure, but was not turbid. The cell count was 68. Globulin was positive with both ammonium sulphate and Noguchi. Differential count (Alzheimer method): Lymphocytes, 0.83; endothelial cells, 0.12; polymorphonuclears, 0.5; plasma cells, 0.

Though the patient did not rally under treatment, he lived until 6 a. m., September 20, when both heart and respiration failed.

This case would be difficult to differentiate from the other two, without the aid of a spinal fluid examination, but with the differential cell count it must be put down as an anterior poliomyelitis (acute ascending type).

I regret that the family physician did not heed my request for a necropsy in this case.

The pathology of acute poliomyelitis being familiar to you, I am offering the following schematic differentiation of three types of acute ascending paralysis, as exemplified by the three cases above reported.

RELATION TO HEMATOMYELIA AND HEMORRHAGIC ENCEPHALITIS

While there is little chance of confusing as striking a case of hemorrhagic myelitis as Case 1 with a typical case of spontaneous hematomyelia, the clinical and pathologic relation of the two should be mentioned, especially as there have been many cases reported in which the clinical symptoms would leave one in doubt whether the process was inflammatory, hemorrhagic or both. Important clinical points to differentiate hematomyelia from acute diffuse hemorrhagic myelitis are: the absence of fever, the suddenness of the onset without premonitory symptoms, often with acute pain, the rapid sequence of paralysis localized in the part corresponding to the hemorrhage, localized anesthesia, lack of progressive extension to new areas, normal white count and normal or nearly normal, spinal fluid constituents.

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TABLE 1.—DIFFERENTIAL DIAGNOSIS

	Ascend. Hem. Myelitis	Landry's Paralysis	Poliomyelitis
Onset	Sudden constitutional symptoms. High fever. Severe pains in legs.	Mild prodromata. Paresthesias. Little or no fever. Pain absent.	Sudden constitutional symptoms. Fever early. Headaches, backaches.
Course	Short duration after hem. occurs. Continuous fever. Complete sensory and motor paralysis, ascending type. Peripheral nerves not involved. Cranial nerves not involved. Sphincters paralyzed. Respiratory failure.	Duration may be prolonged. Fever absent. Motor paralysis complete, only slight sensory. Asc. type. Peripheral nerves occasionally involved. Cranial nerves occasionally involved. Sphincters intact. Respiratory failure.	As paralysis appears, fever begins to subside. Motor paralysis of certain limbs. Mild sensory, or absent. Peripheral nerves seldom involved. Cranial nerves seldom involved. Sphincters occasionally involved. Resp. or heart fails.
Elec. React.	Course too short for abnormalities.	Normal faradic. Normal galvanic..	R. D. develops in paralyzed limbs.
Atrophy	None	None	In paralyzed limbs.
Recovery	Not in typical cases.	Occasionally complete, even after nearly complete paralysis.	In most cases. Atrophy of paralyzed limbs with contractures.
White Count	High	Normal or slight.	High early, slight later.
Spinal Fluid—			
Pressure	Plus	Normal	Plus.
Aspect	Bloody	Normal	Normal.
White Cells	100 +	Under 20	20—100.
Red Cells	Profuse	None	Occasional.
Lymphocytes	Moderate	Occasional	Profuse.
Endothelial Cells	Occasional	Rare	Occasional.
Polymorphonuclears	Profuse	Rare	Rare.
Plasma	Occasional	Rare	Occasional.
Disintegrated	Plus	None	Rare.
Globulin	2 plus	Plus or minus	Plus.

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The pathologic lesions of hematomyelia may be distinguished by their circumscription, the absence of inflammatory products, the picture of blood extravasation transversely, with subsequent ascending and descending degeneration of the tracts, in some cases tubular hemorrhages in the gray matter, and, when not traumatic, intact meninges.

In comparing hemorrhagic myelitis to hemorrhagic encephalitis, or encephalomyelitis, as a pathologic process, certain resemblances appear. For instance, both have the ear marks of an infectious etiology, the hemorrhage being evidently secondary to an inflammatory process in which the meningeal vessels are involved. Strümpell's first case of acute hemorrhagic encephalitis, it will be recalled, followed a bronchopneumonia. An enlarged spleen and other signs of infection were noted. Cultures from the brain, however, were negative. He assumed a primary local infective agent with secondary hemorrhages.

D'Allocco, too, reported a case of acute polioencephalitis in a child, with sudden onset of inflammation symptoms. At necropsy there were vascular occlusions of the cortical vessels following an endophlebitis or endarteritis.

Langbein and Oeller discuss at length the pathology of a case of acute hemorrhagic encephalitis occurring in a woman of 35 years whose acute symptoms lasted only two days. Death occurred one half hour after lumbar puncture. The spinal fluid was under some pressure and was blood-stained. It showed a positive globulin and microscopically a great excess of polymorphonuclear leukocytes but very few lymphocytes. All cultures remained sterile. On section the brain showed areas of thrombosis and hemorrhagic transudation. These authors regarded it as difficult to determine whether the thrombosis was primary or secondary. Because of the infiltration extending to all the vessel coats and even to the connective tissue,

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TABLE 2.—PATHOLOGIC DIFFERENTIATION

	Ascend. Hem. Myelitis	Landry's Paralysis	Poliomyelitis
Spinal Cord—			
Cells	Not degenerated	Degenerated	Degenerated.
Reds	Profuse	None	Few.
Lymphocytes	Increased	Few	Many.
Leukocytes	Many	Absent	Few.
Ameboid glia	Present	Absent	Present.
Endothelial cells	Present	Absent	Present.
Vessels	Engorged and ruptured	Changes slight	Engorged at anterior horns.
White matter	Edematous, infiltrated with hemorrhage. Leukocytes, lymphocytes, etc.	No edema. No hemorrhage. Occasional lymphocytes	No edema. No hemorrhage. No lymphocytes.
Gray matter	Same as white, cells intact or disintegrated	Slight, injected with lymphocytes. Cells degenerated	Marked infiltration with lymphocytes. Cells degenerated.
Blood pigment	Marked	None	Slight.
Lipoid	Cortex	Cord and cortex	Rare.
Periph. Nerves	Normal	Normal, or occasional involvement	Secondary degen. (motor).
Pia—		Slight engorgement	Slight engorgement.
Vessels	Engorged. Edema	No edema	Slight edema.
Infilt. reds	Many	None	Few.
With lymphocytes	Few	Few	Many.
Polymorphonuclears	Many	None	Rare.

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and the presence of meningeal affection, an inflammatory origin would seem likely; the absence of encephalitic changes, however, led the authors to theorize that the extensive arterial oozing may have been due to the thrombotic plugging of the veins. An interesting feature of this case is that the patient had a patent foramen ovale, though no mention was made of clinical phenomena therefrom. The spinal fluid characteristics, with excess of blood and polymorphonuclear cells, are in striking accord with my Case 1.

ETIOLOGY

In the absence of positive proof of the presence of organisms in Case 1, all cultures being negative, and no bacteria found in tissues, we must resort to theoretical grounds for probable etiology.

Barlow, Schiff and several others have reported cases of myelitis consequent to measles, typhoid and other infectious diseases, and I now have under observation a case of localized myelitis following pneumonia. The primary symptoms in Case 1, however, were confined to the spinal cord. Though this girl was generally fatigued, no evidence as to the point of entrance of an infective agent is at hand. That an infectious agent was at work, however, is suggested by the temperature, white count and prostration, which were in contrast with Case 2. The pathologic findings, moreover, bear convincing evidence of an infectious as against a purely toxic reaction, as characterized in Case 2.

In spite of these appearances, it is safe to say that the ordinary infectious organisms are absent, as are, apparently, the ovoid bodies and toxic products of anterior poliomyelitis. At any rate, no characteristic reaction was produced in monkeys by injection of the glycerinated cord from Case 1.

The primary locus of the inflammation in myelitis has led to spirited discussion; whether in the circulatory apparatus as advocated by Mayer and Schmaus,

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the ganglion cells (Charcot), or the glia tissue (Goldscheider, Marinesco). While the evidence in our case points to the primary involvement of the blood vessels, especially as the nerve elements are practically intact except where overwhelmed by the hemorrhage, it must be admitted that in those areas the destruction of all three tissues is fairly complete. Leyden observes that primary hemorrhages of the spinal cord are exceedingly rare, except those due to concussion, and concludes that, anatomically, a sharp separation between primary hemorrhage with subsequent myelitis from primary hemorrhagic myelitis is only possible in the rarest cases.

Circulatory factors affecting the occurrence or extent of hemorrhage must have some consideration. For instance, the permeability of the vessel coats must be dependent on the individual resistance of those coats to the disintegrating power of the toxic products. Another factor that needs consideration is the liquefaction or non-coagulability of the toxic blood. Still another is forced on us by Case 1, namely, the open foramen ovale. We can little more than speculate as to the impaired resistance of the imperfectly oxygenated blood and the abnormal duties which fall on the capillaries thereby, but it cannot be disregarded as a factor, after its occurrence in this striking case and the case of Langbein and Oeller above referred to.

Transmissibility.—It may seem unnecessary to discuss the transmissibility of hemorrhagic myelitis when the agent is unknown, but it may be helpful to draw a contrast on an etiologic basis between the three cases reported above. Assuming that Case 3 was a fulminating case of poliomyelitis, the success of Flexner, Noguchi and others in direct transmission of the disease to monkeys, would warrant the expectation of a similar result from this boy, had his spinal cord been obtainable. On the other hand, the glycerinated cord from Case 1 produced no lesion in monkeys, and, so

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far as this evidence goes, the condition is not similarly transmissible.

The foregoing data likewise are of some importance in making a differential diagnosis between the three types of ascending paralysis, since Case 1 has failed experimentally to produce symptoms of poliomyelitis in monkeys, while Case 2 is typical of ascending toxic paralysis (Landry) from no case of which, so far as I can ascertain, have the globoid bodies been developed or poliomyelitis caused in monkeys.

In this connection I might state that, in a recent personal communication, Dr. Flexner states that he is unaware that any positive infections of monkeys have occurred from cases of typical Landry's paralysis.

It may be remembered that Dr. Gordinier reported before the American Neurological Association last year a case of Landry's type; the spinal cord was then in the hands of Dr. Noguchi for inoculation. Evidently the inoculation in this case was unsuccessful for a recent communication from Dr. Noguchi fails to throw any light on the transmissibility of this disease.

The procedure which to me appears to give the greatest aid in practical differentiation of the three types is the spinal fluid examination, especially the cell count by the Alzheimer method.

Of course, the mere appearance of a fluid which is sanguinolent, that is, uniformly mixed with blood and not coagulated, indicates the hemorrhagic type, but the presence of an enormous preponderance of polymorphonuclear leukocytes over lymphocytes is an effective differentiation from either the Landry's or the poliomyelitic type. The differential count is even more necessary as between Landry's and poliomyelitis, for the gross appearance may be identical and the globulin test is apt to be present in both, but any marked pleocytosis, especially of lymphocytes, with traces of blood or endothelial cells is a strong indica-

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tion against Landry's type and in favor of acute poliomyelitis.

The comment of Dr. Flexner, who, with his staff, was good enough to examine pathologic specimens from this case as well as to inoculate monkeys with the glycerinated cord, was as follows:

"I have examined the specimens and find the lesions as described by your pathologist. They are not typical of poliomyelitis. The only feature in which they resemble poliomyelitis is the infiltration about the vessels in the pia and in the gray matter; but the infiltrations there are relatively slight for the degree of hemorrhage and the cells in some places are chiefly polymorphonuclear and not lymphocytic. On the other hand, the nerve cells do not show the usual necrosis with invasion of leukocytes — or neurophages.

"The inoculations of monkeys with the glycerinated cord were unsuccessful. This does not necessarily mean that the specimen was not derived from a case of poliomyelitis, since it is not possible to transfer all specimens of glycerinated cord from cases of poliomyelitis to monkeys; but taken together with the nontypical character of the lesions I am inclined to think that the case was probably not one of epidemic poliomyelitis."

While it was not my intention to enter the arena of discussion regarding the nosology of acute inflammatory conditions occurring to the spinal cord, I find myself interpreting the suggestions which the foregoing cases seem to bring.

On general principles I believe that names should be descriptive of pathologic processes, and this precept has evolved the somewhat unfamiliar title of my paper. No other designation seems fitting to describe Case 1.

When we come to Case 2, its striking resemblance to the original case reported by Landry in 1859 makes us confident that it belongs to this group, so peculiarly free from macroscopic lesions.

The view held by some authors that no case showing the slightest microscopic change from the normal can properly be called Landry's seems to me untenable. In the first place, it ignores the fact that modern laboratory methods and equipment bring to light lesions which would have been undiscovered not so

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many years ago. Further, it is reasonable to suppose that any etiologic factor virulent enough to cause death must be productive of pathologic changes, whether or not they escape the microscope. These changes naturally become more marked, too, according to the non-resistance of the victim's tissues.

It seems that we should rely more on the type of tissue changes which accompany the clinical data and determine whether they appear to be toxic or grossly inflammatory.

In going over the literature I am not unmindful of the confusion which seems to exist in the minds of many as to the meaning of Landry's paralysis, and, in a measure, I sympathize with the accumulated opinion of Wickman, Taylor, Schmaus and others that the advisability of retaining the title is questionable. Unless, however, these or other authors supply the proof that Landry's paralysis and anterior poliomyelitis are identical it seems our duty to remember the type even though we forget the title.

The suggestion of Taylor to include practically all inflammatory conditions of cord and brain in the term "poliencephalomyelitis" appeals through its convenience, even as the all-inclusive term "psychoneurosis" in functional disorders, but if we all gather under one roof let us erect partitions in their proper places lest we sleep in the kitchen.

By continued animal experimentation and toxicologic research these myelitic diseases must yield up their true etiology and take their corners. Our pride in the work of Flexner, Noguchi and others should make us bent on furnishing them glycerinated material, especially from Landry's type of paralysis, at the earliest opportunity, that our knowledge may increase. Until the time, however, that our laboratories can pin a label on the factors responsible for the three types above reported, I trust it has not been amiss to call attention to their apparent differentiation.

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SUMMARY

An acute ascending hemorrhagic myelitis, as exemplified by Case 1, seems to occupy a unique place allied to, but not identical with, either myelitis or hematomyelia. Its clinical resemblance to acute ascending paralysis, either of the poliomyelitic or Landry's type, is striking, though closer analysis of symptoms and pathologic findings points to the existence, by the clinical data before us, of three distinct types of acute ascending paralysis.

Further confirmation of these types must wait on definite laboratory differentiation, which seems every day to be nearer completion.

In conclusion, I take this opportunity to thank my colleagues for their cooperation in these various cases, and acknowledge the services and valued opinion of Dr. Flexner and his assistants in connection with Case 1. Particularly am I grateful to Dr. M. E. Morse, now of the Boston State Hospital, for her painstaking microscopic work in Cases 1 and 2.

25 High Street.

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ABSTRACT OF DISCUSSION

Dr. Charles R. Ball, St. Paul: In my own work I have been somewhat uncertain as to the etiology of the cases grouped under the heading Landry's paralysis. Whether there is an ascending paralysis of an entirely different origin than the one caused by an acute poliomyelitis seems not as yet to have been proved. In spinal fluid examinations it is very important to differentiate between hemorrhages due to perforations of the blood vessels made at the time of the puncture and hemorrhage from some other cause. Centrifuging the fluid is perhaps the best means of making this differentiation. In recent hemorrhage, after centrifuging, the spinal fluid becomes perfectly clear; in old hemorrhages,

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however, it always shows a yellow tinge due to the presence of the hematin in it. It seems to me that being absolutely certain as to whether the hemorrhage was caused by an accident in puncture, or by the disease itself was one of the most important points to be established in determining the diagnosis in Dr. Burley's cases.

Dr. Benjamin T. Burley, Worcester, Mass.: I quite agree with Dr. Ball that it is very important to get spinal fluid without traumatic blood. It is usually fairly readily told whether the blood is traumatic or not, because usually the traumatic puncture shows blood at first and becomes clearer later. There is a typical appearance to blood which is thoroughly mixed with a spinal fluid and with increase of cells, for which some one seems to have coined the word sanguinolent. Unless one gets a satisfactory puncture the first time, it is advisable to repeat the puncture later.

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